

09/ 891,671

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NEWS 8 Apr 22 Federal Research in Progress (FEDRIP) now available
NEWS 9 Jun 03 New e-mail delivery for search results now available
NEWS 10 Jun 10 MEDLINE Reload
NEWS 11 Jun 10 PCTFULL has been reloaded
NEWS 12 Jul 02 FOREGE no longer contains STANDARDS file segment
NEWS 13 Jul 22 USAN to be reloaded July 28, 2002;
saved answer sets no longer valid
NEWS 14 Jul 29 Enhanced polymer searching in REGISTRY
NEWS 15 Jul 30 NETFIRST to be removed from STN
NEWS 16 Aug 08 CANCERLIT reload
NEWS 17 Aug 08 PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18 Aug 08 NTIS has been reloaded and enhanced
NEWS 19 Aug 09 JAPIO to be reloaded August 25, 2002
NEWS 20 Aug 19 Aquatic Toxicity Information Retrieval (AQUIRE)
now available on STN
NEWS 21 Aug 19 IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 22 Aug 19 The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 23 Aug 26 Sequence searching in REGISTRY enhanced

NEWS EXPRESS February 1 CURRENT WINDOWS VERSION IS V6.0d,
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AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002

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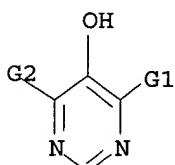
Calculated physical property data is now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details.

registry file, for complete details:
<http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf>

=>
Uploading 09891671.str

L1 STRUCTURE UPLOADED

=> d 11
L1 HAS NO ANSWERS
L1 STR



G1 H, N

G2 H, Ak

Structure attributes must be viewed using STN Express query preparation.

=> s 11
SAMPLE SEARCH INITIATED 17:23:16 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 4046 TO ITERATE

24.7% PROCESSED 1000 ITERATIONS 3 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
PROJECTION ANSWERS: 33 TO 451
PROJECTED ITERATIONS: 77108 TO 84732
PROJECTED ANSWERS: 33 TO 451

L2 3 SEA SSS SAM L1

09/ 891,671

=> s 11 ful
FULL SEARCH INITIATED 17:23:21 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 81672 TO ITERATE

100.0% PROCESSED 81672 ITERATIONS 330 ANSWERS
SEARCH TIME: 00.00.12

L3 330 SEA SSS FUL L1

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COST IN U.S. DOLLARS SINCE FILE TOTAL
FULL ESTIMATED COST ENTRY SESSION
140.28 140.49

FILE 'CAPLUS' ENTERED AT 17:23:39 ON 02 SEP 2002
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FILE COVERS 1907 - 2 Sep 2002 VOL 137 ISS 10
FILE LAST UPDATED: 1 Sep 2002 (20020901/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> s 13
L4 323 L3

=> s electron donating
1115493 ELECTRON
13528 DONATING
L5 9814 ELECTRON DONATING
(ELECTRON (W) DONATING)

=> s 14 and 15
L6 1 L4 AND L5

=> d 16 ibib abs fhitstr

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:472692 CAPLUS
DOCUMENT NUMBER: 101:72692
TITLE: Syntheses of some pyrimidine N-oxides
AUTHOR(S): Jovanovic, Misa V.
CORPORATE SOURCE: Dep. Chem., South. Methodist Univ., Dallas, TX, 75275,
USA
SOURCE: Can. J. Chem. (1984), 62(6), 1176-80

CODEN: CJCHAG; ISSN: 0008-4042

DOCUMENT TYPE:

Journal

LANGUAGE:

English

OTHER SOURCE(S):

CASREACT 101:72692

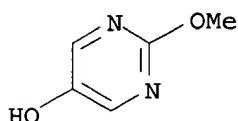
AB Various monosubstituted pyrimidines and methylpyrimidines were N-oxidized with a no. of different peracids. In general, they are more susceptible to side reactions accompanying N-oxidn. than other π -deficient diazines and triazines. Unsym. pyrimidines, which can potentially yield 2 isomeric products, were N-oxidized preferentially at the site para to strong electron-donating substituents. Weaker ring-activating groups, such as Me, are mainly ortho-directing and only aid in the N-oxidn. of pyrimidine nuclei having ortho/para-directing substituents.

IT 91233-70-4P

RL: FORM (Formation, nonpreparative); PREP (Preparation)
(formation of, in N-oxidn. of pyrimidine analogs)

RN 91233-70-4 CAPLUS

CN 5-Pyrimidinol, 2-methoxy- (9CI) (CA INDEX NAME)



=> s inhibit? and oxidation

1503154 INHIBIT?

363690 OXIDATION

L7 24310 INHIBIT? AND OXIDATION

=> d his

(FILE 'HOME' ENTERED AT 17:22:47 ON 02 SEP 2002)

FILE 'REGISTRY' ENTERED AT 17:22:55 ON 02 SEP 2002

L1 STRUCTURE UPLOADED

L2 3 S L1

L3 330 S L1 FUL

FILE 'CAPLUS' ENTERED AT 17:23:39 ON 02 SEP 2002

L4 323 S L3

L5 9814 S ELECTRON DONATING

L6 1 S L4 AND L5

L7 24310 S INHIBIT? AND OXIDATION

=> s 14 and 17

L8 0 L4 AND L7

=> s 17 and pyrimidin?

62774 PYRIMIDIN?

L9 73 L7 AND PYRIMIDIN?

=> s 19 and hydroxy

307940 HYDROXY

L10 14 L9 AND HYDROXY

=> s 17 and (hydroxy same pyrimidin?)

307940 HYDROXY

1176079 SAME

62774 PYRIMIDIN?

0 HYDROXY SAME PYRIMIDIN?

(HYDROXY (W) SAME (W) PYRIMIDIN?)

L11 0 L7 AND (HYDROXY SAME PYRIMIDIN?)

=> s 17 and (hydroxy pyrimidin?)
 307940 HYDROXY
 62774 PYRIMIDIN?
 102 HYDROXY PYRIMIDIN?
 (HYDROXY (W) PYRIMIDIN?)
 L12 0 L7 AND (HYDROXY PYRIMIDIN?)

=> d 19 1- ibib abs fhitstr
 YOU HAVE REQUESTED DATA FROM 73 ANSWERS - CONTINUE? Y/ (N) :y

L9 ANSWER 1 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:858580 CAPLUS
 DOCUMENT NUMBER: 136:145186
 TITLE: Comparison of DNA damage photoinduced by ketoprofen,
 fenofibric acid and benzophenone via electron and
 energy transfer
 AUTHOR(S): Lhiaubet, Virginie; Paillous, Nicole; Chouini-Lalanne,
 Nadia
 CORPORATE SOURCE: Laboratoire des Interactions Moleculaires et
 Reactivite Chimique et Photochimique, Universite Paul
 Sabatier, Toulouse, Fr.
 SOURCE: Photochemistry and Photobiology (2001), 74(5), 670-678
 CODEN: PHCBAP; ISSN: 0031-8655
 PUBLISHER: American Society for Photobiology
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Ketoprofen (KP) and fenofibrate, resp., anti-inflammatory and
 hypolipidemic agents, promote abnormal photosensitivity in patients and may
 induce photoallergic cross-reactions correlated to their benzophenone-like
 structure. Here, their ability to photosensitize the degrdn. of biol.
 targets was investigated in DNA. The photosensitization of DNA damage by
 KP and fenofibric acid (FB), the main metabolite of fenofibrate, and their
 parent compd., benzophenone (BZ), was examd. on a 32P-end-labeled
 synthetic oligonucleotide in phosphate-buffered soln. using gel sequencing
 expts. Upon irradn. at λ > 320 nm, piperidine-sensitive lesions
 were induced in single-stranded oligonucleotides by KP, FB and BZ at all G
 sites to the same extent. This pattern of damage, enhanced in D2O is
 characteristic of a Type-II mechanism. Spin trapping expts. using
 2,2,6,6-tetramethyl-4-piperidone have confirmed the prodn. of singlet
 oxygen during drug photolysis. On double-stranded oligonucleotides,
 highly specific DNA break occurred selectively at 5'-G of a 5'-GG-3'
 sequence, after alkali treatment. Prolonged irradn. led to the degrdn. of
 all G residues, with efficiency decreasing in the order 5'-GG > 5'-GA >
 5'-GC > 5'-GT, in good agreement with the calcd. lowest ionization
 potentials of stacked nucleobase models supporting the assumption of a
 Type-I mechanism involving electron transfer, also obsd. to a lesser
 extent with adenine. Cytosine sites were also affected but the action of
 mannitol which selectively inhibited cytosine lesions suggests,
 in this case, the involvement of hydroxyl radical, also detected by
 electronic paramagnetic resonance using 5,5-dimethyl-1-pyrrolidine-1-oxide
 as spin trap. On a double-stranded 32P-end-labeled 25-mer oligonucleotide
 contg. TT and TTT sequences, the three compds. were found to
 photosensitize by triplet-triplet energy transfer the formation of
 cyclobutane thymine dimers detected using T4 endonuclease V.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:394691 CAPLUS
 DOCUMENT NUMBER: 135:166578

TITLE: Mechanistic investigations of **oxidation** of purine and **pyrimidine** base components of nucleic acids by bromamine-B in aqueous alkaline medium: A kinetic approach
 AUTHOR(S): Vaz, Nirmala; Puttaswamy
 CORPORATE SOURCE: Department of Chemistry, Central College, Bangalore University, Bangalore, 560 001, India
 SOURCE: Studies in Surface Science and Catalysis (2001), 133 (Reaction Kinetics and the Development and Operation of Catalytic Processes), 495-500
 CODEN: SSCTDM; ISSN: 0167-2991
 PUBLISHER: Elsevier Science B.V.
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Mechanism of oxidn. of purine bases (adenine and guanine) and **pyrimidine** bases (uracil, thymine and cytosine) in presence of NaOH by bromamine-B(BAB) was studied. The reactions follow identical kinetics for all the bases, being first order dependence on [BAB]₀ and fractional order each in [substrate]₀ and [NaOH]. Addn. of the reaction product retards the rate and the dielec. effect is pos. Variation of ionic strength and addn. of halide ions had no effect on the rate. Proton inventory studies were made in H₂O-D₂O mixts. for adenine and cytosine. Oxidn. products were identified and activation parameters were evaluated. An isokinetic relation is obsd. with $\beta = 336$ K indicated that enthalpy factors control the rate. The rate of oxidn. of purines is in the order: guanine > adenine while in case of **pyrimidines** the order is thymine > uracil > cytosine. A suitable mechanism is proposed and discussed.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:279860 CAPLUS
 DOCUMENT NUMBER: 135:62958
 TITLE: *Wk* The Chemical Development of CI-972 and CI-1000: A Continuous Nitration, A MgCl₂/Et₃N-Mediated C-Alkylation of a Chloronitropyrimidine, A Catalytic Protodediazotization of a Diazonium Salt, and an Air Oxidation of an Amine
 AUTHOR(S): De Jong, Randall L.; Davidson, James G.; Dozeman, Gary J.; Fiore, Philip J.; Giri, Punam; Kelly, Margaret E.; Puls, Timothy P.; Seamans, Ronald E.
 CORPORATE SOURCE: Pfizer Global Research and Development Holland Laboratories, Holland, MI, 49424, USA
 SOURCE: Organic Process Research & Development (2001), 5 (3), 216-225
 CODEN: OPRDFK; ISSN: 1083-6160
 PUBLISHER: American Chemical Society
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Efficient, large-scale processes were developed for the prepn. of the potent PNP **inhibitors**: 2,6-diamino-3,5-dihydro-7-(3-thienylmethyl)-4H-pyrrolo[3,2-d]**pyrimidin-4-one** hydrochloride monohydrate and 2-amino-3,5-dihydro-7-(3-thienylmethyl)-4H-pyrrolo[3,2-d]**pyrimidin-4-one** hydrochloride monohydrate (I). We report (1) a safe, continuous nitration process for the prepn. of 2-amino-6-chloro-5-nitro-4-**pyrimidinol** and its stable diisopropylamine salt, (2) the first MgCl₂/Et₃N-mediated C-alkylation of a chloronitropyrimidine, (3) a rare catalytic protodediazotization of 2-amino-4-oxo-7-thiophen-3-ylmethyl-4,5-dihydro-3H-pyrrolo[3,2-d]**pyrimidine-6-diazonium chloride**, (4) a single-step process to prep. I directly from 2-amino-6-hydroxy-5-nitro-.alpha.-(3-thienylmethyl)-4-**pyrimidineacetonitrile** using a sponge nickel-catalyzed redn.,

and (5) a method to convert the over-redn. byproduct: 2,5-diamino-6-(1-aminomethyl-2-thiophen-3-yl-ethyl)-pyrimidin-4-ol into I using air oxidn.

REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:107651 CAPLUS
 DOCUMENT NUMBER: 134:279593
 TITLE: Synthesis of chiral pharmaceutical intermediates by oxidoreductases
 AUTHOR(S): Patel, Ramesh N.; Hanson, Ronald L.
 CORPORATE SOURCE: Department of Enzyme Technology, Process Research, Bristol-Myers Squibb Pharmaceutical Research Institute, New Brunswick, NJ, 08903, USA
 SOURCE: ACS Symposium Series (2001), 776 (Applied Biocatalysis in Specialty Chemicals and Pharmaceuticals), 216-247
 CODEN: ACSMC8; ISSN: 0097-6156
 PUBLISHER: American Chemical Society
 DOCUMENT TYPE: Journal; General Review
 LANGUAGE: English

AB A review with 77 refs. Chiral intermediates were prep'd. by enzymic process using oxidoreductases for the chem. synthesis of pharmaceutical drug candidates. These includes: the microbial redn. of 1-(4-fluorophenyl)-4-[4-(5-fluoro-2-pyrimidinyl)-1-piperazinyl]-1-butanone to R-(+)-1-(4-fluoro-phenyl)-4-[4-(5-fluoro-2-pyrimidinyl)-1-piperazinyl]-1-butanol [R-(+)-BMY 14802], an antipsychotic agent; the redn. of N-(4-(1-oxo-2-chloroacetyl ethyl) Ph methane) sulfonamide to corresponding chiral alc., an intermediate for D-(+)-N-[4-[1-Hydroxy-2-[-(methyl ethyl)amino]ethyl]phenyl] methanesulfonamide [D-(+) sotalol], a .beta.-blocker with class III antiarrhythmic properties; biotransformation of N-.epsilon.-carbobenzoxy (CBZ)-L-lysine 7 to CBZ-L-oxylysine, an intermediate needed for synthesis of (S)-1-[6-amino-2-[(hydroxy(4-phenylbutyl) phosphinyl)oxy]1-oxohexyl]-L-proline [ceronapril], a new angiotensin converting enzyme [ACE] inhibitor; enzymic synthesis L-.beta.-hydroxyvaline from .alpha.-keto-.beta.-hydroxy isovalerate. L-.beta.-Hydroxy valine is a key chiral intermediate needed for the synthesis of [S-(Z)]-[[1-(2-Amino-4-thiazolyl)-2-[[2,2-dimethyl-4-oxo-1-(sulfoxy)-3-azetidinyl] amino]-2-oxoethylidene] amino]oxy]acetic acid [tigemonam], a orally active monobactam, enzymic synthesis of L-6-hydroxynorleucine, and enzymic synthesis of (S)-2-amino-5-(1,3-dioxolan-2-yl)-pentanoic acid (allysine ethylene acetal), one of three building blocks used for an alternative synthesis of omapatrilat, a vasopeptidase inhibitor.

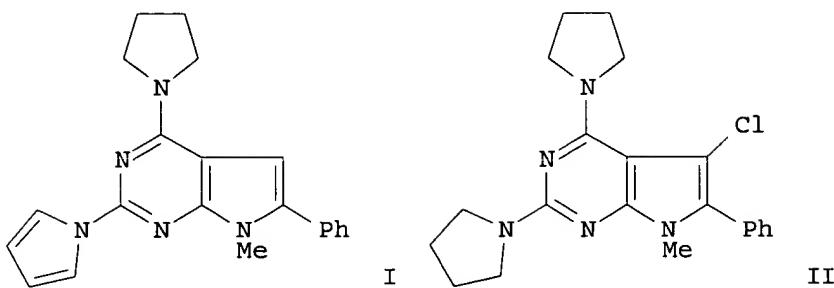
REFERENCE COUNT: 77 THERE ARE 77 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 5 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1998:648906 CAPLUS
 DOCUMENT NUMBER: 130:20319
 TITLE: Structural and functional comparison of agents interfering with dihydroorotate, succinate and NADH oxidation of rat liver mitochondria
 AUTHOR(S): Jockel, Johannes; Wendt, Bernd; Loffler, Monika
 CORPORATE SOURCE: Institute for Physiological Chemistry, School of Medicine, Philipps-University, Marburg, D-35033, Germany
 SOURCE: Biochemical Pharmacology (1998), 56(8), 1053-1060
 CODEN: BCPA6; ISSN: 0006-2952
 PUBLISHER: Elsevier Science Inc.
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Mitochondrially bound dihydroorotate dehydrogenase (EC 1.3.99.11)

catalyzes the fourth sequential step in the de novo synthesis of uridine monophosphate; this enzyme uses ubiquinone as the proximal and cytochrome oxidase as is the ultimate electron transfer system. Here, seven compds. with proven antiproliferative activity and in vitro antipyrimidine effects were investigated with isolated functional mitochondria of rat tissues in order to differentiate their anti-dihydroorotate dehydrogenase potency vs. putative effects on the respiratory chain enzymes. Ten .mu.M of brequinar sodium, the leflunomide derivs. A77-1726, [2-cyano-3-cyclopropyl-3-hydroxy-enoic acid (4-trifluoromethyl)-amide], MNA 279, (2-cyano-N-(4-cyanophenyl)-3-cyclopropyl-3-oxo-propanamide), MNA715 (2-cyano-3-hydroxy-N-4-(trifluoromethyl)-phenyl-6-heptanamide), HR325 (2-cyano-3-cyclopropyl-3-hydroxy-N-[3'-methyl-4'-(trifluoromethyl)phenyl]-propanamide), and the diazine toltrazuril completely **inhibited** the dihydroorotate-induced oxygen consumption of liver mitochondria. Succinate and NADH oxidn. were found to be influenced only at elevated drug concn. (100 .mu.M), with the exception of HR325, 10 .mu.M of which caused a 70% **inhibition** of NADH and 50% **inhibition** of succinate oxidn. This was comparable to the effects of toltrazuril, which caused an approx. 75% **inhibition** of NADH oxidn. Ciprofloxacin was shown here to have only marginal effects on the redox activities of the inner mitochondrial membrane. This differentiation of drug effects on mitochondrial functions will contribute to a better understanding of the in vivo pharmacol. activity of these drugs, which are presently in clin. trials because of their immunosuppressive, cytostatic or anti-parasitic activity. A comparison of the influence of A77-1726, HR325, brequinar and 2,4-dinitrophenol on energetically coupled rat liver mitochondria revealed only a weak uncoupling potential of A77-1726 and brequinar. In addn., a modeling study was raised to search for common spatial arrangements of functional groups essential for binding of **inhibitors** to dihydroorotate dehydrogenase. From the structural comparison of different metabolites and **inhibitors** of **pyrimidine** metab., a 6-point model was obtained by conformational anal. for the drugs tested on mitochondrial functions, pharmacophoric perception and mapping. We propose our model in combination with kinetic data for a rational design of highly specific **inhibitors** of dihydroorotate dehydrogenase.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1998:622209 CAPLUS
 DOCUMENT NUMBER: 129:343459
 TITLE: Chemical Oxidation of 2,4-Diaminopyrrolo[2,3-d]pyrimidines
 AUTHOR(S): Bundy, Gordon L.; Gremban, Robert S.; Banitt, Lee S.; Palmer, John R.; Miszak, Stephen A.; Han, Fusen
 CORPORATE SOURCE: Medicinal Chemistry and Structural Analytical and Medicinal Chemistry, Pharmacia Upjohn Company, Kalamazoo, MI, 49001-0199, USA
 SOURCE: Journal of Organic Chemistry (1998), 63(21), 7542-7546
 CODEN: JOCEAH; ISSN: 0022-3263
 PUBLISHER: American Chemical Society
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI



AB Oxidn. of lipophilic antioxidant PNU-87663 by a variety of nonbiol. oxidizing agents was investigated. E.g., stirring CHCl₃ solns. of PNU-87663 in air for 1 wk gave small amts. of I and II. None of the oxidn. products retained significant levels of lipid peroxidn. inhibiting activity.

L9 ANSWER 7 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:621121 CAPLUS

DOCUMENT NUMBER: 129:239916

TITLE: Therapeutic augmentation of oxyalkylene diesters and butyric acid derivatives with **inhibitors** of fatty acid β -oxidation

INVENTOR(S) : Rephaeli, Ada

PATENT ASSIGNEE(S): Beacon Laboratories, L.L.C., USA

SOURCE: PCT Int. Appl., 58 pp.

CODEN: PIXXD2

DOCUMENT TYPE: **Patent**

LANGUAGE:

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO

WO 9840078	A1	19980917	WO 1998-US4652	19980311
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5939455	A	19990817	US 1997-814222	19970311
AU 9865478	A1	19980929	AU 1998-65478	19980311
ILITY APPLN. INFO.:			US 1997-814222	19970311
			WO 1998-US4652	19980311

AB This invention provides a method of augmenting the therapeutic activity of an oxyalkylene-contg. compd., butyric acid, a butyric acid salt or butyric acid deriv. by administering an **inhibitor** of .beta.-oxidn. of fatty acids to a patient or to host cells. Pharmaceutical compns. are also included.

L9 ANSWER 8 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:186491 CAPLUS

DOCUMENT NUMBER: 128:239464

TITLE: Determination of prodrugs metabolizable by the liver and therapeutic use thereof

INVENTOR(S) : Cheng, Yung-Chi; Chang, Chien-Neng

PATENT ASSIGNEE(S) : Yale University, USA

09/ 891,671

SOURCE: U.S., 24 pp., Cont.-in-part of U.S. Ser. No. 701,462, abandoned.
CODEN: USXXAM

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5728684	A	19980317	US 1994-146164	19940419
ZA 9203495	A	19930331	ZA 1992-3495	19920514
WO 9220816	A1	19921126	WO 1992-US4142	19920515
W: AT, AU, BB, BG, BR, CA, CH, CS, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO				
RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GN, GR, IT, LU, MC, ML, MR, NL, SE				
IL 121375	A1	19981206	IL 1992-121375	19920515
PRIORITY APPLN. INFO.:				
US 1991-701462 B2 19910515				
US 1992-829474 B2 19920203				
WO 1992-US4142 W 19920515				
IL 1992-101879 A3 19920515				

OTHER SOURCE(S): MARPAT 128:239464

AB A method of ascertaining if a prodrug is useful for treating a disease is disclosed. The prodrug is acceptable if it is metabolized in liver cells by aldehyde oxidase to produce an active drug or metabolite. Prodrugs are shown equally effective in treating diseases as the active drug itself with many benefits and without as many assocd. side effects. Methods for treating cancers with e.g. 5-iodo-2-pyrimidinone-deoxyribose are also described.

L9 ANSWER 9 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:126655 CAPLUS

DOCUMENT NUMBER: 128:192666

TITLE: Preparation of acetamides, their use as chymase inhibitors and angiotensin II inhibitors, and cardiovascular agents containing them

INVENTOR(S): Akaha, Atsushi; Takenaka, Kohei; Itani, Hiromichi; Sato, Akihiro; Nakanishi, Isao

PATENT ASSIGNEE(S): Fujisawa Pharmaceutical Co., Ltd., Japan

SOURCE: Jpn. Kokai Tokkyo Koho, 12 pp.

CODEN: JKXXAF

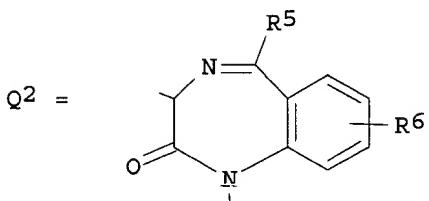
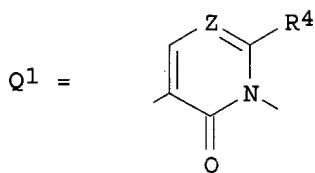
DOCUMENT TYPE: Patent

LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10053579	A2	19980224	JP 1997-160803	19970618
PRIORITY APPLN. INFO.:				
AU 1996-626 19960624				
OTHER SOURCE(S): MARPAT 128:192666				
GI				



AB R1NHXYCONHCHR2COR3 I [R1 = H, protecting group; R2 = ar(lower)alkyl; R3 = lower haloalkyl, (protected) CO2H; X = Q1, Q2; R4, R5 = halo-, lower alkoxy-, or Ph-substituted aryl, cyclo(lower)alkyl; R6 = H, lower alkyl; Z = N, CH; Y = lower alkylene] or their salts, useful for prevention or treatment of heart and/or circulation disorders, are prep'd. by oxidn. of R1aNHXYCONHCHR2CHR3OH (R1a = protecting group; R2, R3, X, Y = same as above) or their salts, followed by optional deprotection. Oxidn. of 905 mg 2-[5-[(benzyloxycarbonyl)amino]-2-(4-fluorophenyl)-1,6-dihydro-6-oxo-1-pyrimidinyl]-N-[2-(4,4,4-trifluoro-3-hydroxy-1-phenyl)butyl]acetamide with 1,1,1-triacetoxy-1,1-dihydro-1,2-benziodoxol-1-(1H)-one at room temp. for 15 h in CH2Cl2 gave 644 mg the corresponding ketone deriv., which inhibited chymase at IC50 of <1.0 .times. 10-5 M.

L9 ANSWER 10 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1997:44786 CAPLUS

DOCUMENT NUMBER: 126:63808

TITLE: Aqueous bath with organosilanes and **oxidation inhibitors** for treatment of copper surface to promote laminate bonding and soldering

INVENTOR(S): Aoyama, Masayuki; Morita, Ryoji; Kawaguchi, Jyun

PATENT ASSIGNEE(S): Henkel Corporation, USA; Aoyama, Masayuki; Morita, Ryoji; Kawaguchi, Jyun

SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9636747	A1	19961121	WO 1996-US6549	19960514
W: CA, US				
JP 08311658	A2	19961126	JP 1995-142656	19950517
US 5925174	A	19990720	US 1997-930080	19971114
PRIORITY APPLN. INFO.:			JP 1995-142656	19950517
			WO 1996-US6549	19960514

OTHER SOURCE(S): MARPAT 126:63808

AB The aq. bath for coating treatment of Cu or cu-alloy surface contains: (a) dissolved and/or dispersed org. solvent; (b) an organosilane coupling agent having functional vinyl, mercapto, amino, and/or glycidyloxy

moieties; and (c) Cu-oxidn. inhibitor selected from azoles, azines, arom. secondary amines, and/or arom. diacyl hydrazide compds. The aq. baths contain the org. solvent at 0.01-15, organosilane at 0.01-30, and the inhibitor at 0.01-5% with the organosilane/inhibitor ratio of (2-8):1, and are suitable for the treatment of Cu foils to improve laminate bonding and soldering for elec. printed circuits. The cleaned Cu foils 35 .mu.m thick can be dip coated for 20 s in the aq. bath contg. 3-(glycidyloxy)propyltrimethoxysilane 0.08, 2-methylimidazole 0.02, and MeOH 10%, followed by drying with hot air at 100.degree.. The treatment provides resistance to Cu migration in close-spaced elec. printed circuits, resistance to thermal damage by molten solder, and increased adhesion strength (esp. at .apprx.150.degree.) in laminates.

L9 ANSWER 11 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1996:629081 CAPLUS

DOCUMENT NUMBER: 125:267783

TITLE: Potent peroxisome proliferators inhibit .beta.-oxidation in the isolated perfused rat liver

AUTHOR(S): Bojes, Heidi K.; Thurman, Ronald G.

CORPORATE SOURCE: Dept. Pharmacolo., University North Carolina Chapel Hill, NC, 27599-7365, USA

SOURCE: Toxicology and Applied Pharmacology (1996), 140(2), 322-327

CODEN: TXAPA9; ISSN: 0041-008X

PUBLISHER: Academic

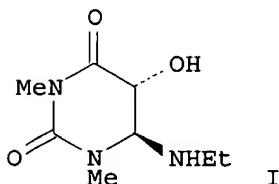
DOCUMENT TYPE: Journal

LANGUAGE: English

AB It is unknown whether peroxisome proliferators decrease hepatic fatty acid oxidn. via uncoupling of respiration or if they inhibit extramitochondrial fatty acyl CoA synthesis. Therefore, the purpose of this study was to examine both processes simultaneously using the isolated perfused liver, a whole cell prepn. where enzymes and biochem. processes can be monitored continuously under nearly physiol. conditions. Accordingly, ketone body formation (.beta.-hydroxybutyrate + acetoacetate) from lipid metab. and oxygen uptake, which is increased by uncoupling agents, were monitored at the same time. 2-Bromooctanoate, a known inhibitor of acyl CoA synthetase, decreased ketone body formation in a dose-dependent manner without altering cellular respiration (half-maximal inhibition, .apprx.25 .mu.M) and concomitantly increased protein kinase C nearly fourfold also in a dose-dependent fashion. Ketogenesis was also blocked maximally 50-66% with mono(ethylhexyl) phthalate, 4-chloro-6-(2,3-xylidino)-2-pyrimidinylthioacetic acid (WY-14,643), and nafenopin, potent peroxisome proliferators and tumor promoters. These compds. also increased protein kinase C three- to fourfold without altering oxygen uptake significantly. Thus, lipid metab. appears to be the prime target of potent peroxisome proliferators most likely on actions via acyl CoA synthetase rather than oxidative phosphorylation. In contrast, weak peroxisome proliferators and tumor promoters, di(ethylhexyl) phthalate and 2-ethylhexanol, did not affect ketogenesis, oxygen consumption, or protein kinase C at similar concns. Addnl., octanoate increased ketone body formation in the presence of nafenopin. Because octanoate is metabolized by mitochondrial acyl CoA synthetase independent of carnitine acyltransferase, these results indicate that the nafenopin does not inhibit mitochondrial .beta.-oxidn. Take together, it is concluded that potent peroxisome proliferators preferentially block ketogenesis without altering cellular respiration in the liver. This phenomenon, which occurs due to inhibition of acyl CoA synthetase, leads to an elevation of free fatty acids that stimulates protein kinase C and promotes cell proliferation.

L9 ANSWER 12 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1996:210224 CAPLUS
 DOCUMENT NUMBER: 124:306408
 TITLE: Effect of various potential **inhibitors**,
 activators and inducers on the N-oxidation
 of isomeric aromatic diazines in vitro using rabbit
 liver microsomal preparations
 AUTHOR(S): Altuntas, T. G.; Gorrod, J. W.
 CORPORATE SOURCE: Chelsea Dep. of Pharmacy, Univ. of London, London, SW3
 6LX, UK
 SOURCE: Xenobiotica (1996), 26(1), 9-15
 CODEN: XENOBH; ISSN: 0049-8254
 PUBLISHER: Taylor & Francis
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB 1. The effects of various potential **inhibitors**, activators and
 inducers on the N-oxidn. of isomeric arom. diazines (pyrazine,
pyrimidine and pyridazine) by rabbit liver microsomes have been
 studied. 2. 2,4-Dichloro-6-phenylphenoxyethylamine (DPEA), SKF 525-A and
 N-octylamine decreased N-oxide formation at 10-4M concns. 3. Methimazole
 and carbon monoxide **inhibited** the N-oxidn. of all three
 substrates studied. 4. The **inhibitory** effects were generally
 exaggerated when hepatic microsomal prepns. from phenobarbitone-pretreated
 animals were used as enzyme source. 5. When phenobarbitone or pyridine
 were used as inducing agents, the N-oxidn. of isomeric arom. diazines
 showed considerable induction, whereas β -naphthoflavone and Aroclor
 1254 pretreatment had much weaker effects. 6. It is suggested that P
 4502E1 and/or 2B are the major subfamilies of P 450 involved in the
 N-oxidn. of isomeric diazines.

L9 ANSWER 13 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1995:689228 CAPLUS
 DOCUMENT NUMBER: 123:340717
 TITLE: Studies on the chemistry of **pyrimidine**
 derivatives with dimethyldioxirane: synthesis,
 cytotoxic effect and antiviral activity of new
 5,6-oxiranyl-5,6-dihydro and 5-hydroxy-5,6-dihydro-6-
 substituted uracil derivatives and **pyrimidine**
 nucleosides
 AUTHOR(S): Saladino, Raffaele; Bernini, Roberta; Crestini,
 Claudia; Mincione, Enrico; Bergamini, Alberto; Marini,
 Stefano; Palamara, Anna Teresa
 CORPORATE SOURCE: Dip. Agrochim. Agrobiol., Univ. Viterbo "La Toscia",
 Viterbo, 01100, Italy
 SOURCE: Tetrahedron (1995), 51(27), 7561-78
 CODEN: TETRAB; ISSN: 0040-4020
 PUBLISHER: Pergamon
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI



AB The oxidn. of uracil derivs. and **pyrimidine** nucleoside performed

in CH₂Cl₂ with dimethyldioxirane afforded new 5,6-oxiranyl-5,6-dihydro and cis-/trans-5,6-dihydroxy-5,6-dihydro-derivs. When the oxidns. were performed in the presence of methanol as nucleophile cis- and trans-5-hydroxy-6-methoxy-5,6-dihydro derivs. were obtained in acceptable yields. Cis- and trans-1,3-dimethyl-5-hydroxy-6-alkylamino-5,6-dihydro uracils were obtained by nucleophilic ring opening of the 1,3-dimethyl-5,6-oxiranyl-5,6-dihydro uracil in the purified form. Interestingly some of the new title products revealed low cytotoxicity and selective antiviral activity against DNA and RNA Viruses. In particular, compd. I shows a strong and selective **inhibition** of the Sendai virus with lower effect on Herpes Simplex-1 virus. Compd. I is also able to slightly **inhibit** HIV-1 virus at high concns., but in this case a cytotoxic effect was obsd.

L9 ANSWER 14 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1993:502416 CAPLUS

DOCUMENT NUMBER: 119:102416

TITLE: Quaternary polyamines as sulfite **oxidation inhibitors** in amine scrubbing of sulfur dioxide

INVENTOR(S): Bedell, Stephen A.

PATENT ASSIGNEE(S): Dow Chemical Co., USA

SOURCE: U.S., 12 pp. Cont.-in-part of U.S. 5,019,365.
CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5167941	A	19921201	US 1990-623313	19901206
US 5019365	A	19910528	US 1990-546075	19900629
PRIORITY APPLN. INFO.:			US 1988-277159	19881129
			US 1990-546075	19900629

OTHER SOURCE(S): MARPAT 119:102416

AB SO₃2- oxidn. is **inhibited** in alk. scrubbing solns. for removal of SO₂ from flue gases by adding 1-3000 ppm of a polyelectrolyte contg. quaternary ammonium groups (mol. wt. >10,000) to the scrubbing soln. The scrubbing soln. contains amines, e.g., piperazinones, morpholinones, piperidines, piperazines, piperazinediones, hydantoins, triazinones, pyrimidones, oxazolidones, and N-carboxymethyl ethylenediamines. Suitable polyelectrolytes include the reaction products of starch and chlorohydroxypropyl tri-Me ammonium salt or glycidyl tri-Me ammonium chloride, poly(diallyldimethylammonium chloride) and copolymers of acrylamide and quaternary ammonium compds.

L9 ANSWER 15 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1991:404696 CAPLUS

DOCUMENT NUMBER: 115:4696

TITLE: Method for making synthetic oligonucleotides which bind specifically to target sites on duplex DNA molecules, by forming a colinear triplex, the synthetic oligonucleotides and methods of use

INVENTOR(S): Hogan, Michael Edward; Kessler, Donald Joseph

PATENT ASSIGNEE(S): Baylor College of Medicine, USA

SOURCE: Eur. Pat. Appl., 40 pp.

CODEN: EPXXDW

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 375408	A1	19900627	EP 1989-313391	19891220
EP 375408	B1	19950222		
R: GR				
CA 2006008	AA	19900620	CA 1989-2006008	19891219
WO 9006934	A1	19900628	WO 1989-US5769	19891220
W: AU, DK, JP				
RW: AT, BE, CH, DE, ES, FR, GB, IT, LU, NL, SE				
AU 9048384	A1	19900710	AU 1990-48384	19891220
AU 640910	B2	19930909		
EP 449972	A1	19911009	EP 1990-901460	19891220
R: AT, BE, CH, DE, ES, FR, GB, IT, LI, LU, NL, SE				
JP 04502407	T2	19920507	JP 1990-502252	19891220
ES 2069598	T3	19950516	ES 1989-313391	19891220
US 5176996	A	19930105	US 1989-453532	19891222
DK 9101200	A	19910620	DK 1991-1200	19910620
PRIORITY APPLN. INFO.:			US 1988-287359	19881220
			WO 1989-US5769	19891220

AB A method for making synthetic oligonucleotides which bind to target sequences in a duplex DNA forming colinear triplexes by binding to the major groove is disclosed. The method includes scanning genomic duplex DNA and identifying nucleotide target sequences .gtoreq.20 nucleotides having either .gtoreq.65% purine bases or .gtoreq.65% **pyrimidine** bases; and synthesizing synthetic oligonucleotides complementary to identified target sequences. The synthetic oligonucleotides have a G when the complementary location in the DNA duplex has a GC base pair and have a T when the complementary location in the DNA duplex has an AT base pair. The synthetic oligonucleotides are oriented 5' to 3' and bind parallel or 3' to 5' and bind antiparallel to the .gtoreq.65% purine strand. Also described are synthetic oligonucleotides made by the above methods. The oligonucleotides can be altered by modifying and/or changing the bases, adding linkers and modifying groups to the 5' and/or 3' termini, and changing the backbone. These synthetic oligonucleotides bind to duplex DNA to form triplexes. This process alters the functioning of the genes which are bound, and can be used to **inhibit** cell growth, alter protein ratios, treat diseases including cancer, and permanently alter the DNA. Oligonucleotides 3'-GTTTTGGGTGTTGTGGGTGTGGTT-5' (HIV29par) and 5'-GTTTTGGGTGTTGTGGGTGTGGTTG-3' (HIV31 anti), designed to bind within the major groove of the DNA helix and form triplexes with specific sequences in the tar region of the human immunodeficiency virus 1 (HIV-1) provirus were readily taken up by HIV-1 infected cells and selectively suppressed synthesis of HIV-1 mRNA without concomitant suppression of mRNA for a constituent gene of the cells. **Inhibition** of viral mRNA was dependent on the dose of oligonucleotide added; max. **inhibition** occurred at 10 mM.

L9 ANSWER 16 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1991:159260 CAPLUS
 DOCUMENT NUMBER: 114:159260
 TITLE: Insertion of specific bases during DNA synthesis past the **oxidation**-damaged base 8-oxodG
 AUTHOR(S): Shibutani, Shinya; Takeshita, Masaru; Grollman, Arthur P.
 CORPORATE SOURCE: Dep. Pharm. Sci., State Univ. New York, Stony Brook, NY, 11794-8651, USA
 SOURCE: Nature (London) (1991), 349(6308), 431-4
 CODEN: NATUAS; ISSN: 0028-0836
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Oxidative damage to DNA, reflected in the formation of 8-oxo-7-hydrodeoxyguanosine (8-oxodG), may be important in mutagenesis, carcinogenesis and the ageing process. Y. Kuchino et al. (1987) studied

DNA synthesis on oligodeoxynucleotide templates contg. 8-oxodG, concluding that the modified base lacked base pairing specificity and directed misreading of **pyrimidine** residues neighboring the lesion. The present study reports different results, using an approach in which the several products of a DNA polymerase reaction can be measured. In contrast to the earlier report, it was found that dCMP and dAMP are incorporated selectively opposite 8-oxodG with transient **inhibition** of chain extension occurring 3' to the modified base. The potentially mutagenic insertion of dAMP is targeted exclusively to the site of the lesion. The ratio of dCMP to dAMP incorporated varies, depending on the DNA polymerase involved. Chain extension from the dA.cntdot.8-oxodG pair was efficiently catalyzed by all polymerases tested.

L9 ANSWER 17 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1989:511633 CAPLUS
 DOCUMENT NUMBER: 111:111633
 TITLE: Factors affecting the photooxidation of purine and **pyrimidine** bases sensitized by hypocrellin A
 AUTHOR(S): Jia, Hongti; Dong, Cangyu; Wu, Yaonan
 CORPORATE SOURCE: Dep. Biochem., Beijing Med. Univ., Beijing, Peop. Rep. China
 SOURCE: Shengwu Huaxue Zazhi (1989), 5(3), 275-80
 CODEN: SHZAE4; ISSN: 1000-8543
 DOCUMENT TYPE: Journal
 LANGUAGE: Chinese
 AB Studies of hypocrellin A-sensitized photooxidn. of purine and **pyrimidine** bases were carried out using optical absorption and HPLC techniques. The UV absorbance of guanine and thymine in the presence of hypocrellin A (3 times. 10-5M) at pH 9.0 decreased significantly after visible light irradn. for 40 min. A new peak occurred in the reversed-phase HPLC elution profile for guanine photooxidn. sensitized by hypocrellin A. The new peak had an absorption max. at 475 nm. It is proposed that the product(s) formed from hypocrellin A-sensitized photooxidn. of guanine might be cleavage product(s) of the purine ring. The rate of hypocrellin A-sensitized photooxidn. of guanine was dependent on pH, illumination time, and sensitizer concn. Azide, a specific quencher for 102, could partially inhibit the photooxidn. of guanine at a concn. of 40 mM and completely at concns. >110 mM. Thus, 102 is involved in hypocrellin A-sensitized photooxidn. of guanine. The possible photooxidn. pathway of guanine was discussed.

L9 ANSWER 18 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1989:226853 CAPLUS
 DOCUMENT NUMBER: 110:226853
 TITLE: Inhibition of autoxidation of divicine and isouramil by the combination of superoxide dismutase and reduced glutathione
 AUTHOR(S): Winterbourn, Christine C.
 CORPORATE SOURCE: Christchurch Sch. Med., Christchurch Hosp., Christchurch, N. Z.
 SOURCE: Arch. Biochem. Biophys. (1989), 271(2), 447-55
 CODEN: ABBIA4; ISSN: 0003-9861
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The effects of GSH on the autoxidn. of the fava bean **pyrimidine** aglycons, divicine and isouramil, and on acid-hydrolyzed vicine (provisional identification 2-amino-4,5,6-trihydroxypyrimidine) have been studied. GSH alone promoted redox cycling of each compd., with concomitant GSH oxidn. and H2O2 prodn. In the presence of superoxide dismutase, there is a lag period during which little **pyrimidine** oxidn. occurs, followed by a period of accelerated oxidn. With the three **pyrimidines**, increasing concns. of GSH extended this lag period

and progressively decreased subsequent rates of both **pyrimidine** oxidn. and O uptake. No GSH oxidn. or O uptake occurred during the lag. These results show that the combination of GSH and superoxide dismutase is able to **inhibit** redox cycling of the **pyrimidines**. With a 10-fold excess of GSH over isouramil or acid-hydrolyzed vicine (20-fold with divicine) this coupled oxidn. of GSH and the **pyrimidine** is almost completely suppressed. This mechanism may be a means whereby GSH in combination with superoxide dismutase protects against the cytotoxic effects of these reactive **pyrimidines**.

L9 ANSWER 19 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1989:149326 CAPLUS

DOCUMENT NUMBER: 110:149326

TITLE: **Auto-oxidation** of dialuric acid, divicine and isouramil. Superoxide dependent and independent mechanisms

AUTHOR(S): Winterbourne, Christine C.; Cowden, William B.; Sutton, Harry C.

CORPORATE SOURCE: Christchurch Sch. Med., Christchurch Hosp., Christchurch, N. Z.

SOURCE: Biochem. Pharmacol. (1989), 38(4), 611-18
CODEN: BCPCA6; ISSN: 0006-2952

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The toxicity of dialuric acid to pancreatic .beta. cells, and the hemolytic action of divicine and isouramil involve auto-oxidn. and redox cycling reactions. Divicine and isouramil are produced on hydrolysis of the fava bean glycosides, vicine and convicine. The mechanism of auto-oxidn. of the 3 compds. as well as the acid hydrolysis product of vicine (provisionally assigned the structure 2-amino-4,5,6-trihydroxypyrimidine) has been studied. All 4 **pyrimidines** auto-oxidized rapidly at neutral pH, generating H2O2 by an O-dependent chain mechanism. Superoxide dismutase-**inhibited** lag period varied with pH, temp., and **pyrimidine** concn., and was much shorter for isouramil and divicine than for dialuric acid and acid-hydrolyzed vicine. The initial rate of dialuric acid oxidn. was greater and the acceleration less pronounced than with the other **pyrimidines**. A mechanism common to all 4 **pyrimidines** has been shown by kinetic anal. to account for nearly all the observations in the presence and absence of superoxide dismutase. Autocatalysis in the latter case is attributed mainly to the reactions reduced **pyrimidine** + oxidized **pyrimidine** .dblharw. 2 **pyrimidine** radical and **pyrimidine** radical + O2 .fwdarw. oxidized **pyrimidine** + O2-. Rate consts. for these and other reactions are reported. At pH 7.4 and 37.degree., the lag period before 100 .mu.M acid-hydrolyzed vicine underwent rapid oxidn. was .apprx.15 min. Isouramil and divicine at an equiv. concn. gave lags of <1 min, which became less at higher concns. Thus, intracellular superoxide dismutase should provide only transitory protection against the oxidn. products of dialuric acid, divicine, or isouramil. Prolonged protection should only be achieved if the accumulation of oxidized **pyrimidine** is also prevented.

L9 ANSWER 20 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1987:593866 CAPLUS

DOCUMENT NUMBER: 107:193866

TITLE: The **oxidation** of 4-**pyrimidinone** and 4-quinazolinone and their N-methyl derivatives by milk xanthine oxidase

AUTHOR(S): Bunting, John W.; Luscher, Mark A.; Redman, Jane

CORPORATE SOURCE: Dep. Chem., Univ. Toronto, Toronto, ON, M5S 1A1, Can.
SOURCE: Bioorg. Chem. (1987), 15(2), 125-40

CODEN: BOCMBM; ISSN: 0045-2068

DOCUMENT TYPE: Journal
 LANGUAGE: English
 OTHER SOURCE(S): CASREACT 107:193866
 AB 4-Pyrimidinone, 4-quinazolinone, and each of their N1-Me derivs. are oxidized to the corresponding 2,4-diones by milk xanthine oxidase. Steady-state kinetic parameters were evaluated for the enzymic oxidn. of these substrates over the pH range 5.0-10.5. The pH dependences of k_{cat}/K_m (k_{cat} = catalytic const.) for 4-pyrimidinone and 4-quinazolinone are consistent with the neutral mols. of these species being substrates, but their anionic conjugate bases not being enzymically oxidized. Apart from this substrate ionization, k_{cat} and K_m do not show any dramatic pH dependence. 1-Ethyl-4-pyrimidinone is slowly oxidized by this enzyme, and 3-methyl-4-pyrimidinone is an extremely poor substrate; 3-methyl-4-quinazolinone is not enzymically oxidized. These latter 2 species are competitive inhibitors for the oxidn. of 4-pyrimidinone. The 2- and 4-pyrimidinones, the 2- and 4-quinolinones, 1-isoguinolinone, and each of their N-Me derivs. were reversible inhibitors for this enzyme and ID₅₀ values (concns. giving half-maximal inhibition) values were evaluated. These data are consistent with the neutral 1H tautomers of 4-pyrimidinone and 4-quinazolinone being the true substrates for this enzyme. The low reactivity of 4-quinazolinone as a substrate can probably be traced to reversible inhibition by 4(3H)-quinazolinone of the enzymic oxidn. of 4(1H)-quinazolinone.

L9 ANSWER 21 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1985:199779 CAPLUS
 DOCUMENT NUMBER: 102:199779
 TITLE: Site-specific DNA damage caused by lipid peroxidation products
 AUTHOR(S): Ueda, Kazumitsu; Kobayashi, So; Morita, Junji; Komano, Tohru
 CORPORATE SOURCE: Dep. Agric. Chem., Kyoto Univ., Kyoto, 606, Japan
 SOURCE: Biochim. Biophys. Acta (1985), 824(4), 341-8
 CODEN: BBACAO; ISSN: 0006-3002
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB DNA damage induced by autoxidized lipids was investigated using covalently closed, circular (supercoiled) DNA and DNA fragments of defined sequence. DNA strand-breaking substances accumulated during autoxidn. of Me linolenate, and strand breakage was measured with samples taken at different times. The DNA strand-breaking activity reached its max. a little after the peak value of peroxide and decreased upon further autoxidn. The peak of the DNA strand-breaking activity did not always coincide with the peak of thiobarbituric acid reactants or of conjugated diene, either. The DNA strand-breaking reaction was dependent on metal ions and was inhibited by KI and tiron and partially by catalase, suggesting the involvement of radical species and (or) O₂ radicals. No direct cleavage of singly end-labeled 100-200 base-pair DNA fragments by autoxidized Me linolenate and Cu²⁺ was detected under the conditions used. Cleavage occurred during subsequent heating in piperidine after the reaction. The alkali-labile damage was preferentially induced at pyrimidine residues, esp. in dinucleotide sequences of pyrimidine-guanine (5'-fwdarw.3'), as detd. by sequencing.

L9 ANSWER 22 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1985:45928 CAPLUS
 DOCUMENT NUMBER: 102:45928
 TITLE: 1,3-Oxazolidin-4-one derivatives
 INVENTOR(S): Krepelka, Jiri; Pouzar, Vladimir
 PATENT ASSIGNEE(S): Czech.
 SOURCE: Czech., 2 pp.

AB The sulfoxides I ($R = H$ or a 1-4 C alkyl group, $R' =$ heterocyclic group of arom. character contg. 1 or 2 N atoms selected from pyridin-3-yl (optionally substituted by a 1-4 C (or halogen) contg. group), quinolin-3-yl, pyridazin-4-yl, **pyrimidin-5-yl**, thiazol-5-yl, thieno[2,3-b]pyridin-5-yl and thieno[3,2-b]pyridin-6-yl, and Y is a valency bond or methylene group) are obtained by the electrochem. oxidn. of the ring S atom of a thioformamide deriv. N-Methyl-2-(pyridin-3-yl)tetrahydrothiophen-2-carbothioamide [82081-31-0] is electrochem. oxidized to form trans-N-methyl-2-(pyridin-3-yl)tetrahydrothiophen-2-carbothioamide 1-oxide [92569-64-7].

L9 ANSWER 24 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1983:422835 CAPLUS
 DOCUMENT NUMBER: 99:22835
 TITLE: Pattern of hydroxyl radical addition to cytosine and 1-, 3-, 5-, and 6-substituted cytosines. Electron transfer and dehydration reactions of the hydroxyl adducts
 AUTHOR(S): Hazra, D. K.; Steenken, S.
 CORPORATE SOURCE: Max-Planck-Inst. Strahlenchem., Muelheim, D-4330, Fed. Rep. Ger.
 SOURCE: J. Am. Chem. Soc. (1983), 105(13), 4380-6
 CODEN: JACSAT; ISSN: 0002-7863
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB By use of the technique of pulse radiolysis with optical detection, the isomer distribution of the radicals formed in aq. soln. by addn. of OH radicals to cytosine and its derivs., e.g., 5-methylcytosine, 3-methylcytosine, cytidine, 5-methylcytidine, cytidylic acid and 2'-deoxycytidine, has been detd. by utilizing differences between the isomeric OH adducts with resp. to electron-transfer reactions with the reductant N,N,N',N' -tetramethyl-p-phenylenediamine (TMPD) or the oxidant tetranitromethane (TNM). The radicals of Cy-5-OH, formed by addn. of OH to C(5) of cytosine (87% of the OH radicals), 3- and 5-methylcytosine (92% and 65%, resp.), 5-carboxycytosine (82%), and 2-amino-4-hydroxy-6-methylpyrimidine (95%) reduce TNM to yield nitroform anion. The radicals Cy-6-OH, formed by addn to C(6), oxidize TMPD to TMPD⁺. The Cy-5-OH radicals undergo a base-catalyzed dehydration to yield readicals that are able to oxidize TMPD to TMPD⁺. In the case of cytosine the dehydrated OH adduct is identical with the one-electron oxidn. product from the reaction of cytosine with SO_4^- . If N(1) of the **pyrimidine** ring is substituted as with 1-methylcytosine, cytidine, cytidylic acid, 2'-deoxycytidine and 2'-deoxycytidylic acid, no dehydration of the OH adducts occur. In contrast, substitution by alkyl at N(3) does not inhibit the dehydration of the corresponding Cy-5-OH radical. In the presence of oxygen the Cy-5-OH and Cy-6-OH radicals are converted into peroxy radicals which oxidize TMPD. In basic soln. these peroxy radicals decomp., presumably by elimination of O_2^- . With 5-methylcytosine a peroxy radical derived from the radical formed by H abstraction from the Me group is addnl. obsd. With the cytosine nucleosides and nucleotides the probability of OH attack at the cytosine mol. is >80%.

L9 ANSWER 25 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1982:178179 CAPLUS
 DOCUMENT NUMBER: 96:178179
 TITLE: Comparative effects of ancydrol and its analogs on growth of peas and ent-kaurene oxidation in cell-free extracts of immature *Marah macrocarpus* endosperm
 AUTHOR(S): Coolbaugh, Ronald C.; Swanson, David I.; West, Charles A.
 CORPORATE SOURCE: Dep. Botany, Iowa State Univ., Ames, IA, 50011, USA

09/ 891,671

SOURCE: Plant Physiol. (1982), 69(3), 707-11
CODEN: PLPHAY; ISSN: 0032-0889
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The plant growth retardant .alpha.-cyclopropyl-.alpha.-(4-methoxyphenyl)-5-pyrimidine Me alc. (ancymidol) and a series of its analogs having one or more of the substituents varied, were tested for their comparative biol. activity. The compds. were tested as **inhibitors** of internode elongation in peas and as **inhibitors** of the oxidn. of ent-kaurene catalyzed by microsomal preps. from the liq. endosperm of *M. macrocarpus* seeds. The relative effectiveness of a substance was generally the same as an **inhibitor** of the 2 processes. Ancymidol was the most effective. Substitution of the alc. group of ancymidol by either Me or H groups reduced the activity only slightly. Substitution of the cyclopropyl group by an iso-Pr moiety also had little effect on the activity. However, substitution of the cyclopropyl group with a Ph or other aryl substituent greatly reduced the effectiveness of the analog as an **inhibitor**. Replacement of the 4-methoxyphenyl substituent with a similar substituent such as 4-chlorophenyl had little effect on activity, but replacement with a 2-methoxyphenyl group greatly reduced activity. Analogs in which the pyrimidyl moiety of ancymidol was modified were inactive in whole plants, but moderately active in the cell-free ent-kaurene oxidn. system. The application of gibberellic acid can overcome the growth **inhibitions** caused by treatment of the test plants with $\text{.1 to } \text{req. } 10^{-5}\text{M}$ of the **inhibitors**. However, the **inhibitory** effects of $\text{.1 to } \text{req. } 10^{-4}\text{M}$ **inhibitors** on test plants were not overcome by the applications of exogenous gibberellic acid. The effects of low concns. of these substances on plant growth are primarily a consequence of **inhibition** of ent-kaurene oxidn. and gibberellin biosynthesis. Other modes of **inhibition** may operate at higher **inhibitor** concns.

L9 ANSWER 26 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1982:47794 CAPLUS
DOCUMENT NUMBER: 96:47794
TITLE: Oxidation of pyrimidine and purine deoxyribonucleosides by reactive oxygen species generated by the hydrolytic decomposition of potassium perchromate
AUTHOR(S): Cadet, J.; Balland, A.; Voituriez, L.; Hahn, B. S.; Wang, S. Y.
CORPORATE SOURCE: Lab. Radiobiol., CEN, Grenoble, Fr.
SOURCE: Oxygen Oxy-Radicals Chem. Biol., [Proc. Int. Conf.] (1981), Meeting Date 1980, 610-11. Editor(s): Rodgers, Michael A. J.; Powers, Edward Lawrence.
Academic: New York, N. Y.
CODEN: 46WOAA
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Anal. of the decompn. products resulting from K_3CrO_8 oxidn. of deoxyribothymidine, deoxyribocytosine, deoxyriboadenosine, and deoxyriboguanosine suggested that **pyrimidine** oxidn., which is **inhibited** by the OH.bul. scavenger MeOH, results from OH.bul. addn. to the 5,6-**pyrimidine** bond followed by the fast reaction of O_2 , whereas purine oxidn., which is **inhibited** by the singlet O scavenger N3, is due to singlet O generation. O_2^- which is also produced in the K_3CrO_8 reaction is not reactive toward the nucleosides. Thus, K_3CrO_8 is a convenient oxidizing agent for the selective degrdn. of DNA components either by OH.bul. or single O.

L9 ANSWER 27 OF 73 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1979:518847 CAPLUS
DOCUMENT NUMBER: 91:118847

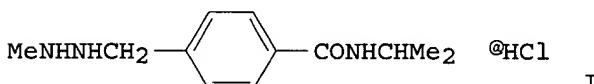
TITLE: Catalytic release of deoxyribonucleic acid bases by oxidation and reduction of an iron .cntdot. bleomycin complex
 AUTHOR(S): Povirk, Lawrence F.
 CORPORATE SOURCE: Dep. Mol. Biophys. Biochem., Yale Univ., New Haven, CT, 06520, USA
 SOURCE: Biochemistry (1979), 18(18), 3989-95
 CODEN: BICHAW; ISSN: 0006-2960
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The kinetics and stoichiometry of several reactions involving bleomycin, Fe, DNA, O, and sulfhydryls were examd. in order to assess their possible role in degrdn. of DNA by bleomycin. Oxidn. of Fe(II) in the presence of bleomycin resulted in an Fe(III).cntdot.bleomycin complex, having an optical absorption spectrum with a broad shoulder at 320-400 nm, which was stable for several hours. If Fe(II) was allowed to oxidize before bleomycin addn., the complex did not form. The complex was reduced by dithiothreitol 5 times faster than unchelated Fe(III), and redn. of the complex was inhibited by high concns. of DNA. However, stopped-flow studies showed that, when sufficient DNA was present to bind most of the Fe(II).cntdot.bleomycin, its rate of oxidn. by O was 60 times faster than that of unbound Fe(II).cntdot.bleomycin. Under the same conditions, oxidn. of each mol of DNA-bound Fe(II).cntdot.bleomycin released 0.18 mol of thymine. Treatment of pyrimidine-labeled Escherichia coli DNA with bleomycin and high concns. of Fe(II) and 2-mercaptoethanol resulted in the release of .1toreq.2.4 mol of pyrimidines (of which 60% were thymine) per mol of bleomycin. This result implies that each Fe.cntdot.bleomycin complex went through several cycles of oxidn. and redn. and that bleomycin usually was not inactivated in the base-release reaction. In supercoiled plasmid pDR3709 DNA, 1 base was released per single-strand break (measured in alkali), eliminating the possibility of multiple base release during a single bleomycin-DNA interaction. Thus, the Fe.cntdot.bleomycin complex acts as a catalyst which, after being reduced by sulfhydryls, binds to DNA in a way which facilitates both the oxidn. of the chelated Fe(II) and the degrdn. of the DNA backbone by the products of this oxidn.

L9 ANSWER 28 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1977:596313 CAPLUS
 DOCUMENT NUMBER: 87:196313
 TITLE: Oxidation of hypoxanthines, bearing 8-aryl or 8-pyridyl substituents, by bovine milk xanthine oxidase
 AUTHOR(S): Bergmann, Felix; Levene, Lawrence; Govrin, Hanna
 CORPORATE SOURCE: Hadassah Med. Sch., Hebrew Univ., Jerusalem, Israel
 SOURCE: Biochim. Biophys. Acta (1977), 484(2), 275-89
 CODEN: BBACAQ
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Hypoxanthines, contg. aryl or pyridyl substituents at position 8, were converted by bovine milk xanthine oxidase into their corresponding xanthines at low rates. Oxidn. was accelerated considerably when the 8-pyridyl substituents were quaternized. In the enzymic oxidn. of quaternary 8-pyridylhypoxanthines, a lag phase preceded the attainment of a const., max. reaction rate. The delay is assumed to be due to a relatively slow conformational change in the active enzymic center. In 8-(3'-N-methylpyridinio)xanthine betaine, the pyridinium moiety was also attacked at high pH (9-11) to yield an N-methyl-2-pyridone. The analogous pyridone was the only oxidn. product of 1-methyl-8-(3'-N-methylpyridinio)hypoxanthine betaine, which was not attacked in the pyrimidine ring. The cationic substrates were attracted to the enzyme by an anionic group, which probably formed an ion pair with a protonated NH₂ group in or near the active center.

L9 ANSWER 29 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1977:67403 CAPLUS
 DOCUMENT NUMBER: 86:67403
 TITLE: Influence of 8-substituents on the **oxidation**
 of hypoxanthine and 6-thioxopurine by bovine milk
 xanthine oxidase
 AUTHOR(S): Bergmann, Felix; Levene, Lawrence; Govrin, Hanna;
 Frank, Aryeh
 CORPORATE SOURCE: Hadassah Med. Sch., Hebrew Univ., Jerusalem, Israel
 SOURCE: Biochim. Biophys. Acta (1977), 480(1), 39-46
 CODEN: BBACAQ
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The effect of 8-substituents on the rate of oxidn. of hypoxanthine and
 6-thioxopurine by bovine milk xanthine oxidase was studied. An 8-Me group
 does not alter the rate of oxidn. of hypoxanthine materially, but an 8-Ph
 substituent reduces it markedly. This is ascribed to **inhibition**
 of the tautomerization process, responsible for substrate activation,
 prior to oxidn. In contrast, the 8-Ph group in 3-methyl-8-
 phenylhypoxanthine enhances the rate, presumably by binding to a
 hydrophobic site near the enzymic center. An 8-Ph group in 6-thioxopurine
 markedly increases the rate of enzymic oxidn. Probably the arom.
 substituent diverts anion formation to the imidazole ring. In contrast,
 ionization of 8-methyl-6-thioxopurine involves the **pyrimidine**
 moiety, thus rendering enzymic attack at position 2 more difficult.

L9 ANSWER 30 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1977:66977 CAPLUS
 DOCUMENT NUMBER: 86:66977
 TITLE: Kinetic study of the DNA-natulan reaction mechanism.
 Part III. NMR investigation of the effect of
 nucleotides on natulan **oxidation**
 AUTHOR(S): Belova, L. A.; Zenin, S. V.; Emanuel, N. M.
 CORPORATE SOURCE: Chem. Fac., Moscow State Univ., Moscow, USSR
 SOURCE: Stud. Biophys. (1976), 60(3), 171-9
 CODEN: STBIBN
 DOCUMENT TYPE: Journal
 LANGUAGE: Russian
 GI



AB The autoxidn. of natulan (I) in the presence and absence of ribonucleoside
 monophosphates was analyzed by ¹H NMR in phosphate buffer in D₂O. During
 autoxidn. in the absence of nucleotides, the intensities of the resonance
 lines for the NMe, Me₂CH, and CH₂ groups of I decreased, and that for the
 C₆H₆ ring H atoms disappeared and was replaced by a broad unresolved band.
 Two new resonance lines also appeared that were assigned to NMe groups of
 reaction products. Thus, autoxidn. of I may initially consist of the
 cleavage of its CH₂-NH band. Similar changes were obsd. during the
 autoxidn. of I in the presence of nucleotides. However, the rate of
 autoxidn. of 0.1M I was significantly decreased by 0.2M AMP; moderately
 decreased by 0.2M CMP, 0.1M rTMP, and(or) 0.2M UMP; and slightly decreased
 by 0.02M GMP. At equiv. concns., GMP and AMP may exhibit similar
inhibitory effects. The purine nucleotides may exhibit higher
inhibitory activities than the **pyrimidine** nucleotides as
 the result of the increased ability of the former to form complexes with

I. The presence of nucleotides stabilized a reaction product that was identified as methylhydrazine. Thus, I can undergo autoxidn. in nucleotide complexes and, possibly, also in DNA complexes. The subsequent hydrolysis of the hydrazine may cause the cleavage of the DNA chain which was previously obsd. in DNA-natulan solns.

L9 ANSWER 31 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1976:560012 CAPLUS

DOCUMENT NUMBER: 85:160012

TITLE: Fused **pyrimidines. II. Synthesis and oxidation of 3-aminoisothiazolo[3,4-d]pyrimidines**

AUTHOR(S): Furukawa, Yoshiyasu; Shima, Shunsuke

CORPORATE SOURCE: Cent. Res. Div., Takeda Chem. Ind., Ltd., Osaka, Japan

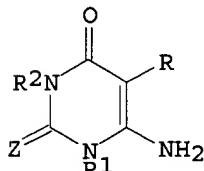
SOURCE: Chem. Pharm. Bull. (1976), 24(5), 979-86

CODEN: CPBTAL

DOCUMENT TYPE: Journal

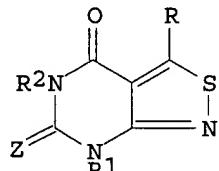
LANGUAGE: English

GI



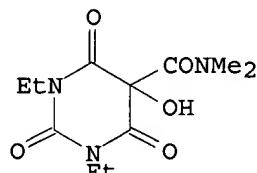
I, R=H, Z=O, S

II, R=C(S)NHR³, Z=O, S



III, R=NHR³

IV, R=NR³R⁴



V

AB The 6-aminouracils I (R1 = PhCH₂, Et, H, Me, p-ClC₆H₄, MeOCH₂CH₂, Ph, Me₂CHCH₂, p-MeOC₆H₄; R2 = H, Et) reacted with SCN₃ (R₃ = Et, Me, C₆H₄Cl-p, Ph, C₆H₄OH-p, CH₂Ph) to give the thiocarbamoyluracils II, which were oxidized with Br or H₂O₂ to give the isothiazolopyrimidinediones III, whose alkylation with R₄I (R₄ = Me, Et) gave the (disubstituted amino)isothiazolopyrimidines IV. Further oxidn. of III or IV (R₁ = R₂ = Et, R₃ = R₄ = Me) with H₂O₂ gave the hydroxybarbituric acid V. Aminoisothiazolopyrimidines are potential cyclic nucleotide phosphodiesterase **inhibitors**.

L9 ANSWER 32 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1976:518637 CAPLUS

DOCUMENT NUMBER: 85:118637

TITLE: **Oxidation of selected pteridine derivatives by mammalian liver xanthine oxidase and aldehyde oxidase**

AUTHOR(S): Hodnett, C. N.; McCormack, J. J.; Sabean, J. A.

CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, Vt., USA

SOURCE: J. Pharm. Sci. (1976), 65(8), 1150-4

CODEN: JPMSAE

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Xanthine oxidase, obtained from rat liver, oxidizes a variety of substituted amino- and hydroxypteridines in a manner identical to that previously obsd. for milk xanthine oxidase. For example, 2-aminopteridine and its 4- and 7-OH derivs. are oxidized efficiently to 2-amino-4,7-dihydropteridine (isoxanthopterin) by the rat liver enzyme and 4-aminopteridine and its 2- and 7-OH derivs. are oxidized to 4-amino-2,7-dihydroxypteridine. 4-Hydroxypteridine and the isomeric 2-

and 7-hydroxypyteridines are oxidized by rat liver xanthine oxidase to 2,4,7-trihydroxypyteridine. Rabbit liver aldehyde oxidase, but not rat liver xanthine oxidase, catalyzes the oxidn. in position 7 of 2,4-diaminopteridine and its 6-Me and 6-hydroxymethyl derivs. 2-Aminopteridine and 4-aminopteridine are both oxidized to the corresponding 7-OH derivs. in the aldehyde oxidase system; 2-amino-4-hydroxypyteridine appears to be a minor product in the **oxidation** of 2-aminopteridine by rabbit liver aldehyde oxidase. Both aldehyde oxidase and xanthine oxidase catalyze the oxidn. of 2-amino-6,7-disubstituted pteridines to the corresponding 4-OH derivs.; 4-hydroxy-6,7-disubstituted pteridines are oxidized in position 2 by both enzymes. 4-Amino-6,7-disubstituted pteridines are not oxidized by either enzyme. 2-Amino-4-methylpteridine is oxidized in position 7 by aldehyde oxidase but is not an effective substrate for xanthine oxidase; 2-hydroxypyteridine and 7-hydroxypyteridine are not oxidized to a detectable extent by aldehyde oxidase. All oxidns. mediated by xanthine oxidase are strongly **inhibited** by allopurinol (4-hydroxypyrazolo[3,4-d]pyrimidine), and all oxidns. mediated by aldehyde oxidase are **inhibited** by menadione (2-methyl-1,4-naphthoquinone). Rat liver xanthine oxidase and, to a lesser extent, rabbit liver aldehyde oxidase are **inhibited** by 4-chloro-6,7-dimethylpteridine; 2-amino-3-pyrazinecarboxylic acid **inhibits** xanthine oxidase but not aldehyde oxidase. The oxidns. of 2- and 4-aminopteridines by aldehyde oxidase results in concomitant redn. of cytochrome c.

L9 ANSWER 33 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1976:174883 CAPLUS
 DOCUMENT NUMBER: 84:174883
 TITLE: Microsomal N-**oxidation** of the
 hepatocarcinogen N-methyl-4-aminoazobenzene and the
 reactivity of N-hydroxy-N-methyl-4-aminoazobenzene
 AUTHOR(S): Kadlubar, Fred F.; Miller, James A.; Miller, Elizabeth
 C.
 CORPORATE SOURCE: Med. Sch., Univ. Wisconsin, Madison, Wis., USA
 SOURCE: Cancer Res. (1976), 36(3), 1196-206
 CODEN: CNREA8
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI

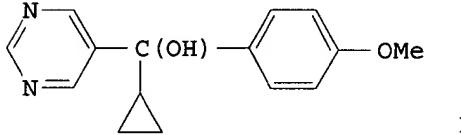


AB The N-oxidn. of N-methyl-4-aminoazobenzene (MAB) (I) [621-90-9] was catalyzed by hepatic microsomes in a reduced pyridine nucleotide- and oxygen-dependent reaction. The initial N-oxidn. product, N-hydroxy-N-methyl-4-aminoazobenzene (N-HO-MAB) [1910-36-7], was readily oxidized to a second product that yielded N-hydroxy-4-aminoazobenzene [6530-27-4] upon subsequent acid treatment. The secondary N-oxidn. product may be formed nonenzymatically and is presumed to be N-HO-MAB N-oxide [58989-03-0] or its dehydrated deriv., N-(p-phenylazophenyl)nitro [58989-01-8]. Under the same conditions, MAB was also oxidatively N-dealkylated to 4-aminoazobenzene [60-09-3], which was N-oxidized to N-hydroxy-4-aminoazobenzene [6530-27-4]. Unlike the latter reactions, the microsomal N-oxidn. of MAB was independent of cytochrome P-450, as shown by its lack of sensitivity to **inhibition** by 2-[(2,4-dichloro-6-phenyl)phenoxy]ethylamine and its inability to utilize cumene hydroperoxide in place of reduced pyridine nucleotides and oxygen. The N-oxidn. of MAB was also catalyzed by the purified microsomal

flavoprotein mixed-function amine oxidase [9059-11-4]. The noncarcinogenic dye N-ethyl-4-aminoazobenzene [2058-67-5] was metabolized similarly to MAB. For male animals the hepatic levels of MAB N-oxidase [59088-29-8] activity were in the order: rat > hamster, guinea pig > mouse, rabbit. Little or no MAB N-oxidase activity was present in several extrahepatic rat tissues. N-HO-MAB, N-hydroxy-N-ethyl-4-aminoazobenzene [58989-02-9], and N-hydroxy-4-aminoazobenzene catalyzed the aerobic oxidn. of cysteine and glutathione. These hydroxylamines also bound covalently to proteins. The binding of N-HO-MAB with nucleic acids was only 3 to 6% that obsd. with serum albumin. Under anhydrous conditions the nitrone generated aerobically from N-HO-MAB reacted with carbon-carbon or carbon-nitrogen double bonds, or both, in fatty acids, retinol, purines, and **pyrimidines** to yield isoxazolidine and/or oxadiazolidine addn. products. The nitrone from N-hydroxy-N-ethyl-4-aminoazobenzene was much less reactive under these conditions. Syntheses of N-HO-MAB and N-hydroxy-N-ethyl-4-aminoazobenzene are reported.

L9 ANSWER 34 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1976:145955 CAPLUS
 DOCUMENT NUMBER: 84:145955
 TITLE: **Inhibition of ent-kaurene oxidation**
 and growth by .alpha.-cyclopropyl-.alpha.- (p-
 methoxyphenyl)-5-**pyrimidine** methyl alcohol
 Coolbaugh, Ronald C.; Hamilton, Roxanne
 Dep. Nat. Sci. Math., Oregon Coll. Educ., Monmouth,
 Oreg., USA
 SOURCE: Plant Physiol. (1976), 57(2), 245-8
 CODEN: PLPHAY
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI



I

AB Growth of Alaska peas (*Pisum sativum*) was **inhibited** >60% by 19.5 and 39 .mu.M ancyimidol (I) [12771-68-5] treatment. This growth **inhibition** was reversed completely by gibberellic acid [77-06-5] application. Cell-free enzyme preps. from pea shoot tips and wild cucumber (*Marah oregana*) endosperm were used to test the effects of this substituted **pyrimidine** on the incorporation of mevalonic acid-14C into ent-kaurene [562-28-7] and ent-kaurenol [2300-11-0], resp. I (10-6M) completely blocked the conversion of ent-kaurene to ent-kaurenol. I at higher concns. (10-3M) **inhibited** the incorporation of mevalonic acid-14C into ent-kaurene less than it did at lower I concns. One mode of action of I is the **inhibition** of gibberellin biosynthesis.

L9 ANSWER 35 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1976:74294 CAPLUS
 DOCUMENT NUMBER: 84:74294
 TITLE: Pyrido[2,3-d]**pyrimidinedione** derivatives
 INVENTOR(S): Noda, Kanji; Nakagawa, Akira; Miyata, Satoru;
 Motomura, Toshiharu; Ide, Hiroyuki
 PATENT ASSIGNEE(S): Hisamitsu Pharmaceutical Co., Inc., Japan
 SOURCE: Japan. Kokai, 6 pp.
 CODEN: JKXXAF

DOCUMENT TYPE: Patent
 LANGUAGE: Japanese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 50100090	A2	19750808	JP 1974-5049	19740105
JP 57061757	B4	19821225		

GI For diagram(s), see printed CA Issue.
 AB **Pyrido[2,3-d]pyrimidinediones** I (R1 = aryl, cycloalkyl, aralkyl; R2 = substituted alkyl, unsatd. alkyl) were prep'd. by oxidn. of II (Z = CO, CS, CH2; X = halo, inorg. or org. ester group). I had analgesic, antiinflammatory, and central nerve depressant activities (no data). Thus, a mixt. of 3 g II (R1 = m-F3CC6H4, R2 = Et, Z = CH2, X = EtSO4), 5 g K2Cr2O7, and 100 ml concd. H2SO4-H2O (1:1) was refluxed 3 hr to give 1.8 g I (R1 = m-F3CC6H4, R2 = Et). Among 152 more I prep'd. were (R1, R2 given): m-BrC6H4, Me; Ph, Me; Ph, Et; and Ph, Pr.

L9 ANSWER 36 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1975:589717 CAPLUS
 DOCUMENT NUMBER: 83:189717
 TITLE: **Oxidation of 7-aminothiadiazolo(3,4-d)pyrimidines and 7-aminofurazano(3,4-d)pyrimidines by xanthine oxidase and aldehyde oxidase**
 AUTHOR(S): McCormack, John J.; Taylor, Edward C.
 CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, Vt., USA
 SOURCE: Biochem. Pharmacol. (1975), 24(17), 1636-9
 CODEN: BCPA6
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI For diagram(s), see printed CA Issue.
 AB Oxidn. of 7-aminothiadiazolo[3,4-d]pyrimidine (I) by xanthine oxidase from milk (Km 5 .times. 10-5M) occurred at the unsubstituted 5 position of the heterocyclic ring, yielding 7-amino-5-hydroxythiadiazolo[3,4-d]pyrimidine. Oxidn. of I by aldehyde oxidase was similar to that by xanthine oxidase, with a Km of 1.2 .times. 10-4M. Oxidn. of 7-aminofurazano[3,4-d]pyrimidine (II) by xanthine oxidase occurred with a Km of 7 .times. 10-5M, and that by aldehyde oxidase with a Km 1.5 .times. 10-4M. The 5-methyl, 5-amino, and 5-methylthio derivs. of II acted neither as substrates nor inhibitors of the enzymes.

L9 ANSWER 37 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1973:463815 CAPLUS
 DOCUMENT NUMBER: 79:63815
 TITLE: **Distribution, subcellular localization, and product inhibition of dihydroorotate oxidation in the rat**
 AUTHOR(S): Kennedy, James
 CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, N.Y., USA
 SOURCE: Arch. Biochem. Biophys. (1973), 157(2), 369-73
 CODEN: ABBIA4
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The enzyme system for converting dihydroorotate to orotate was distributed in all rat tissues assayed. In liver, the reaction is localized in mitochondria and appears to be specific for dihydroorotate. Evidence is presented for excluding the reaction as a site for control of **pyrimidine biosynthesis via end-product inhibition**. However, product inhibition by orotic acid does occur and is

competitive with dihydroorotate.

L9 ANSWER 38 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1973:144682 CAPLUS
 DOCUMENT NUMBER: 78:144682
 TITLE: **Inhibition** of ferritin reduction by
pyrazolo[3,4d]pyrimidines
 AUTHOR(S): Duggan, D. E.; Streeter, K. B.
 CORPORATE SOURCE: Merck Inst. Ther. Res., West Point, Pa., USA
 SOURCE: Arch. Biochem. Biophys. (1973), 156(1), 66-70
 CODEN: ABBIA4
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The characteristics of **inhibition** of the ferritin reductase function of xanthine oxidase by 3 pyrazolopyrimidines is described. Under anaerobic conditions, the ferritin reduction coupled to hypoxanthine **oxidation** was **inhibited** by the 3 pyrazolopyrimidines tested, 4- hydroxyprazoloprimidine, 4,6-dihydroxyprazoloprimidine, and 4- mercaptopyrazolopyrimidine. Under the same conditions, 4 purine analogs were devoid of **inhibitory** activity, and 8-azaguanine and 8-azaadenine were weakly **inhibitory**.

L9 ANSWER 39 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1973:83315 CAPLUS
 DOCUMENT NUMBER: 78:83315
 TITLE: Sulfanilamides as **inhibitors** of
oxidation of ammoniacal nitrogen in soils
 INVENTOR(S): Goya, Hirohito; Nakanishi, Michio; Saruwatori,
 Kenichi; Hirose, Akira; Shinozawa, Tetsuichi
 PATENT ASSIGNEE(S): Yoshitomi Pharmaceutical Industries, Ltd.
 SOURCE: Jpn. Tokkyo Koho, 6 pp.
 CODEN: JAXXAD
 DOCUMENT TYPE: Patent
 LANGUAGE: Japanese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 47004966	B4	19720212	JP 1969-54885	19690711

GI For diagram(s), see printed CA Issue.
 AB Sulfanilamide derivs. (I or II), where X is -H, -COCH₃, -C(:NH)NH₂, 2-pyridinyl, 2-**pyrimidinyl**, and 2-pyrazinyl, R is H or a half amide of a C₂-C₈ dicarboxylic acid and R₁ is 1,2-benzendiyl or (CH₂)_n (n = 0-6), were active as **nitrification inhibitors**. They can be applied together with the fertilizer or after fertilizer application and remain active up to the next growth period.

L9 ANSWER 40 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1973:83310 CAPLUS
 DOCUMENT NUMBER: 78:83310
 TITLE: Sulfanilamide **oxidation inhibitors** for ammoniacal nitrogen in soils
 INVENTOR(S): Goya, Hirohito; Hidaka, Nobuhiro; Hirose, Akira;
 Shinozawa, Tetsuichi
 PATENT ASSIGNEE(S): Mitsui Toatsu Chemicals Co., Ltd.
 SOURCE: Jpn. Tokkyo Koho, 5 pp.
 CODEN: JAXXAD
 DOCUMENT TYPE: Patent
 LANGUAGE: Japanese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 47004961	B4	19720212	JP 1968-22045	19680405

GI For diagram(s), see printed CA Issue.
 AB New sulfanilamide nitrification **inhibitors** were developed; they have a general structure I, where Y may be H, 2-pyridinyl, 4-methyl-2-thiazolyl, 1,3,4-thiadiazolyl, 2-**pyrimidinyl**, amidino, benzoyl, 4,6-dichloro-s-triazin-2-yl, or 4,6-diamino-s-triazin-2-yl. Z is C2-6 alkyl, and R and R1 are H or Me. Good results were obtained with 1-150 ppm **inhibitor** concns. in soil. The **inhibitors** can be applied before, at, or after org. N fertilizer is introduced to the soil and they remain effective also in the next growing season. The **inhibitors** can be applied to the soil surface and then plowed into soil or by spray or as an emulsion.

L9 ANSWER 41 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1973:15263 CAPLUS
 DOCUMENT NUMBER: 78:15263
 TITLE: **Oxidation** of diarylmethylpyridine and **pyrimidine** carbanions. Steric requirements
 AUTHOR(S): Kress, Thomas J.; Moore, Larry L.
 CORPORATE SOURCE: Lilly Res. Lab., Eli Lilly and Co., Indianapolis, Indiana, USA
 SOURCE: J. Heterocycl. Chem. (1972), 9(5), 1161-4
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI For diagram(s), see printed CA Issue.
 AB Oxidn. of 2-,3-, and 4-(diphenylmethyl)pyridine, 5-(diphenylmethyl)-
pyrimidine, and 5-[(2,4-dichlorophenyl)phenylmethyl]
pyrimidine (I; R = Cl, R1 = R2 = R3 = H) by NaOH and O in Me2SO
 gave 72-97% triaryl carbinols. The following I did not react: R = R2 =
 H, R1 = R3 = Cl; R = R1 = Cl, R2 = R3 = H; R = R1 = R3 = Cl, R2 = H; r =
 R1 = R2 = Cl, R3 = H. Thus, the steric shielding imposed on the central C
 atom of the carbanion intermediate by 2 axially opposed Cl atoms
inhibited an effective collision with O.

L9 ANSWER 42 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1972:98388 CAPLUS
 DOCUMENT NUMBER: 76:98388
 TITLE: Oxidations of nucleic acids and their components in soil. I. **Oxidation** in air-dried soil samples
 AUTHOR(S): Drobniкова, Vera
 CORPORATE SOURCE: Dep. Plant Physiol. Soil Biol., Charles Univ., Prague, Czech.
 SOURCE: Zentralbl. Bakteriol., Parasitenk., Infektionskr. Hyg., Abt. 2 (1971), 126(7), 700-12
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The degree of oxidn. of RNA was lower than that of its individual components. With storage, the respiration of air-dried soil increased, but the content of NO3- changed very little. Ribose oxidn. was lower than that of glucose. N and P, each, increased glucose oxidn. and nitrification in soil. NH4)2CO3 or purine or **pyrimidine** bases depressed the respiration rate during the 1st hr even in the presence of glucose. Greater oxidn. occurred with purines than with glucose. Hypoxanthine was oxidized 1st, followed by adenine and uric acid. The oxidn. of **pyrimidines** began earlier but at a lower rate than that of purines. Addn. of glucose shortened the primary oxidn. of purines and **pyrimidines**, but the rate of oxidn. remained unchanged. In the oxidn. of purine and **pyrimidine** ribosides, the sugar moiety

was probably oxidized 1st, followed by oxidn. of the bases.

L9 ANSWER 43 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1971:135921 CAPLUS
 DOCUMENT NUMBER: 74:135921
 TITLE: Xanthine oxidase-mediated oxidation of epinephrine
 AUTHOR(S): Valerino, Donald M.; McCormack, John J.
 CORPORATE SOURCE: Coll. Med., Univ. Vermont, Burlington, Vt., USA
 SOURCE: Biochem. Pharmacol. (1971), 20(1), 47-55
 CODEN: BCPCA6
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI For diagram(s), see printed CA Issue.
 AB Epinephrine bitartrate (I) stimulation of purines oxidn. by xanthine oxidase (XO) was attributable to concomitant oxidn. of I as well as purine substrate, hypoxanthine (II), by XO preps. XO did not oxidize I under the conditions employed in the absence of an oxidizable substrate such as II. The oxidn. of I in the presence of XO and II was inhibited strongly by allopurinol (4-hydroxypyrazolo-(3,4-d)-pyrimidine). The product of I oxidn. in the XO system was adrenochrome (2,3-dihydro-3-hydroxy-N-methylindole-5,6-quinone).

L9 ANSWER 44 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1969:446108 CAPLUS
 DOCUMENT NUMBER: 71:46108
 TITLE: Oxidation of some aminopteridines by xanthine oxidase
 AUTHOR(S): Valerino, Donald M.; McCormack, John J.
 CORPORATE SOURCE: Coll. of Med., Univ. of Vermont, Burlington, Vt., USA
 SOURCE: Biochim. Biophys. Acta (1969), 184(1), 154-63
 CODEN: BBACAQ
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB 2-Aminopteridine and its 4- and 7-monohydroxylated derivs. were efficiently oxidized at pH 7.4 and 37. degree. by milk xanthine oxidase to a product identified as 2-amino-4,7-dihydroxypteridine (isoxanthopterin) on the basis of spectroscopic and chromatographic data. The 7-methyl and the 6,7-dimethyl derivs. of 2-aminopteridine were rapidly oxidized by xanthine oxidase, but 4-methyl-2-aminopteridine did not appear to be an effective substrate for the enzyme. 4-Aminopteridine and its 2- and 7-monohydroxy derivs. were converted into 4-amino-2,7-dihydroxypteridine by xanthine oxidase. 4-Aminopteridine and its 6,7-dimethyl deriv. were oxidized considerably less rapidly than the corresponding 2-aminopteridines. The order of susceptibility of the aminopteridines (2- > 4-) to oxidn. by xanthine oxidase was different from that previously reported by Bergmann and Kwiety for the analogous hydroxypteridines (4- > 2-). 4-Hydroxypyrazolo-(3,4-d)-pyrimidine (allopurinol) strongly inhibited the oxidn. of the aminopteridines by xanthine oxidase.

L9 ANSWER 45 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1969:413086 CAPLUS
 DOCUMENT NUMBER: 71:13086
 TITLE: Role of alkyl substitution in 2,3-disubstituted and 3-substituted 4-quinazolones on the inhibition of pyruvic acid oxidation
 AUTHOR(S): Parmar, Surendra S.; Kishor, K.; Seth, P. K.; Arora, R. C.
 CORPORATE SOURCE: King George's Med. Coll., Lucknow Univ., Lucknow, India
 SOURCE: J. Med. Chem. (1969), 12(1), 138-41
 CODEN: JMCMAR

DOCUMENT TYPE: Journal
 LANGUAGE: English
 GI For diagram(s), see printed CA Issue.
 AB Several 2,3-disubstituted and 3-substituted 4-quinazolones I (R = Me and H; R1 = Et and Me2) were synthesized to investigate structure-activity relation of these quinazolones with respect to their ability to inhibit pyruvic acid oxidn. by rat brain homogenate. 2-Methyl-3-(o-tolyl)-4-quinazolone was used for comparison. In general, 2,3-disubstituted quinazolones exhibited greater inhibitory properties as compared to the corresponding 3-substituted quinazolones. Introduction of the alkyl substituent(s) on the phenyl nucleus, attached to the 3 position of the quinazolone mol., significantly influenced the enzyme inhibitory properties of these quinazolones. In both series, max. inhibition of the oxidn. of pyruvic acid was observed with quinazolones synthesized from 2,4-dimethylaniline. Increase in the concn. of the quinazolones simultaneously increased the enzyme inhibition. Added NAD, responsible for the increase in the respiratory activity of brain homogenate during oxidn. of pyruvic acid, reduced the inhibition produced by 2,3-disubstituted and 3-substituted 4-quinazolones.

L9 ANSWER 46 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1968:424726 CAPLUS
 DOCUMENT NUMBER: 69:24726
 TITLE: Enzymic decomposition of urocanic acid. VII.
 Identification of the enzyme catalyzing the oxidation of 4(5)-imidazolone-5(4)-propionic acid as an aldehyde oxidase
 AUTHOR(S): Payes, Benjamin; Greenberg, David M.
 CORPORATE SOURCE: Sch. of Med., Univ. of California, San Francisco, Calif., USA
 SOURCE: Arch. Biochem. Biophys. (1968), 125(3), 911-17
 CODEN: ABBIA4
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB The enzyme from guinea pig liver that catalyzes the oxidn. of 4(5)-imidazolone-5(4)-propionic acid to hydantoin-5-propionic acid has properties similar to those described for aldehyde oxidase (or so-called xanthine dehydrogenase). The guinea pig liver enzyme catalyzes oxidn. of aldehydes, purines, a pyrimidine, and a no. of other N-contg. heterocyclic compds. The ratio of enzyme activity on imidazolone propionate and 4-hydroxypyrimidine remained reasonably const. over a 130-fold enrichment of enzyme purity. The guinea pig liver enzyme responded similarly to activators and inhibitors of the aldehyde oxidases from other sources. The enzyme resembles the so-called xanthine dehydrogenase in that it catalyzes oxidn. of purine to 8-hydroxypurine, not 6-hydroxypurine. Utilization of O for the oxidn. of hypoxanthine and xanthine is very sluggish, but their oxidn. is markedly accelerated by other electron acceptors, e.g., 2,6-dichlorophenolindophenol, as is the case with xanthine dehydrogenase.

L9 ANSWER 47 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1967:450375 CAPLUS
 DOCUMENT NUMBER: 67:50375
 TITLE: Oxidation of pyrimidine nucleosides and nucleotides by osmium tetroxide
 AUTHOR(S): Burton, Kenneth
 CORPORATE SOURCE: Massachusetts Gen. Hosp., Boston, Mass., USA
 SOURCE: Biochem. J. (1967), 104, 686-94
 CODEN: BIJOAK
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Pyrimidine nucleosides such as thymidine, uridine, or cytidine

are oxidized readily at 0.degree. by osmium tetroxide in NH4Cl buffer. There is virtually no oxidn. in bicarbonate buffer of similar pH. Oxidn. of 1-methyluracil yields 5,6-dihydro-4,5,6-trihydroxy-1-methyl-2-pyrimidone. Osmium tetroxide and ammonia react reversibly in aq. soln. to form a yellow 1:1 complex, probably OsO₃NH. A second mol. of ammonia must be involved in the oxidn. of UMP since the rate of this reaction is approx. proportional to the square of the concn. of unprotonated ammonia. 4-Thiouridine reacts with osmium tetroxide much more rapidly than does uridine. The changes of absorption spectra are different in NaHCO₃ buffer and in NH₄Cl buffer. They occur faster in the latter buffer and, under suitable conditions, cytidine is a major product. Poly(uridylic acid) is oxidized readily by ammoniacal osmium tetroxide, but its oxidn. is inhibited by poly(adenylic acid). Pyrimidines of yeast amino acid-transfer RNA are oxidized more slowly than the corresponding mononucleosides, esp. the thymine residues. Appreciable oxidn. can occur without change of sedimentation coeff.

L9 ANSWER 48 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1967:9261 CAPLUS
 DOCUMENT NUMBER: 66:9261
 TITLE: Possible role of xanthine oxidase in the oxidation of glyoxylate to oxalate
 AUTHOR(S): Gibbs, Dorothy A.; Watts, Richard W. E.
 CORPORATE SOURCE: St. Bartholomew's Hosp., London, Engl.
 SOURCE: Clin. Sci. (1966), 31(2), 285-97
 CODEN: CSCIAE
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Xanthine oxidase catalyzed the oxidn. of glyoxylate to oxalate, but played only a minor role in the overall production of oxalate in the intact human. The Km for milk xanthine oxidase with respect to glyoxalate was 6 times 10⁻⁴M, and the reaction was inhibited by allopurinol (4-hydroxypyrazolo-[3,4-d]pyrimidine) and by pteridylaldehyde (2-hydroxy-4-amino-6-formylpteridine), but not by disulfiram (tetraethylthiuram disulfide). The oxidn. of glyoxylate in the supernatant fraction of human liver tissue was less susceptible to inhibition by allopurinol and pteridylaldehyde than the corresponding fraction from rat tissue. Studies on persons with xanthinuria showed that oxalate excretion was not abolished when xanthine oxidase was congenitally absent. 37 references.

L9 ANSWER 49 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1966:431470 CAPLUS
 DOCUMENT NUMBER: 65:31470
 ORIGINAL REFERENCE NO.: 65:5867g-h
 TITLE: New aspects of the oxidation of glycolate in etiolated plants
 AUTHOR(S): Scoppa, P.; Tafuri, F.
 CORPORATE SOURCE: Univ. Perugia, Italy
 SOURCE: Ann. Fac. Agrar. Univ. Studi, Perugia (1964), 19, 99-121
 DOCUMENT TYPE: Journal
 LANGUAGE: Italian

AB Green wheat (Thatcher variety) plants contain an active glycolic acid oxidase (I), while in etiolated plants I activity is practically negligible. Adding FMN to slurries of green or etiolated plants produces an increase of I activity, a final concn. of 10⁻⁴M being necessary for the max. effect. The addn. of FMN produces an increase of enzyme activity equal to .apprx.10-fold the initial value in etiolated plants and .apprx.2.5-fold in green plants. .alpha.-Hydroxy-2-pyrimidinemethanesulfonate and the hydrazide of isonicotinic acid, inhibitors of I, do not affect activation due to the addn. of FMN to etiolated plants. If glycolate is added to slurries of etiolated

plants, or the slurries are maintained at ambient temps., notable changes in absorbance are observed; this does not happen with slurries of green plants.

L9 ANSWER 50 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1966:430660 CAPLUS
 DOCUMENT NUMBER: 65:30660
 ORIGINAL REFERENCE NO.: 65:5714e-f
 TITLE: Antioxidative effects of purine bases on hydrogen peroxide oxidation of **pyrimidine** bases
 AUTHOR(S): Melzer, M. S.; Tomlinson, R. V.
 CORPORATE SOURCE: State Univ. of New York, Buffalo
 SOURCE: Arch. Biochem. Biophys. (1966), 115(1), 226-9
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Guanylic, polyadenylic, and adenylic acids were tested for their abilities to inhibit H₂O₂ oxidn. of the base moieties of polycytidylic and polyuridylic acids. Guanylic acid protected both of these substrates, but adenylic acid protected neither. Polyadenylic acid showed antioxidative capacity only when complex formation could occur, i.e., in the presence of polyuridylic acid. The chelating agent, EDTA, was as effective as guanylic acid in protecting both **pyrimidine** substrates.

L9 ANSWER 51 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1961:54900 CAPLUS
 DOCUMENT NUMBER: 55:54900
 ORIGINAL REFERENCE NO.: 55:10581g-i,10582a-c
 TITLE: Deoxyribonucleic acid (DNA) synthesis, respiration, and virulence in pneumococci
 AUTHOR(S): Firschein, W.
 CORPORATE SOURCE: Wesleyan Univ., Middletown, CT
 SOURCE: Ann. N.Y. Acad. Sci. (1960), 88, 1054-74
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB Selective stimulation of DNA synthesis occurred in resting cells of type I and type II virulent pneumococci (I) supplemented with enzymic DNA digests, all of the naturally occurring deoxynucleosides and deoxynucleotides, and the 4 nucleoside diphosphates derived from ribonucleic acid (RNA). Cells of the avirulent forms (II) did not respond appreciably to these supplements. Characteristic of the enhanced DNA synthesis with I was a decrease in synthetic activity from the max. levels after 30 or 50 min. A supplement contg. the enzymic DNA digest, Mn⁺⁺, and purines and **pyrimidines** also stimulated DNA synthesis by virulent pneumococci, but the magnitude was not as great as with the nucleoside deriv. supplement; the mechanism of action appeared to be different. The Mn⁺⁺-contg. supplement enhanced DNA, RNA, and protein syntheses; the nucleoside deriv. affected DNA synthesis primarily. The active material from the enzymic DNA digest was not identical with known breakdown products and apparently acted other than as a mere contributor of DNA precursors. The optimum supplementation system also elicited a striking selective stimulation of O uptake in I contg. glucose and inhibited O uptake when II was present. In the absence of glucose I was incapable of oxidizing the nucleic acid breakdown products, whereas cells of II exhibited definite activity. A substantial amt. of the sugar was assimilated in both suspensions when the extracellular supplements were absent. Some of this assimilated glucose was oxidized in suspensions of I when the nucleic breakdown products were added. A substance contg. twice as much N as that found in the entire extracellular nucleic acid breakdown supplement did not enhance O uptake in I to any degree. The deoxynucleotide mixt. and the enzymic DNA digest in combination produced the greatest stimulation of O consumption. The deoxynucleoside and the nucleoside diphosphate mixts. exerted a depressing effect on the max.

levels obtained with these other supplements. Any supplement contg. the enzymic DNA digest stimulated O uptake to a greater extent than such supplements lacking the digest. High concns. of single deoxynucleosides, except for deoxycytidine which **inhibits** respiration, enhanced oxidn. of glucose almost as well as the full supplement. Mn⁺⁺ stimulated glucose oxidn. in I while **inhibiting** such **oxidation** in II.

L9 ANSWER 52 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1959:40050 CAPLUS
 DOCUMENT NUMBER: 53:40050
 ORIGINAL REFERENCE NO.: 53:7216e-h
 TITLE: Derivatives of 6-methyl-1,2,4-triazine
 PATENT ASSIGNEE(S): Wellcome Foundation Ltd.
 DOCUMENT TYPE: Patent
 LANGUAGE: Unavailable
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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GB 802122		19581001	GB	

GI For diagram(s), see printed CA Issue.
 AB The triazines N:CY.CMe:N.N:CX (I), where X may be HO, HS, RS, or RNH, and Y may be HS or H2N may be prep'd. by reactions with P2S5 or NH3, **oxidation** with KMnO4 or alkylation with Me2SO4. Freshly-ground P2S5 (30 g.) and 100 ml. Tetralin were added to 10 g. I (X = Y = OH), the mixt. was stirred at 180-190.degree. for 3 hrs., cooled, filtered, the ppt. washed with petr. ether and decompd. with 250 ml. H2O, and the aq. soln. worked up with ether to give orange plates of I (X = Y = HS) (II), m. 217-18.degree. (boiling H2O). II treated at 122.degree. in a sealed tube with alc. NH3 (satd. at 0.degree.) gave quant. yields of I (X = HS, Y = H2N) (III), beige needles repptd. from cold 0.1N HCl with cold 0.1N NaOH, darkened at 270.degree., did not m. at 310.degree.. III (10 g.) was oxidized with 25 g. KMnO4 in 500 ml. H2O, filtered and the filtrate concd. to dryness. The K sulfonate was dissolved in 20 ml. H2O, brought to pH 1 with 0.1N HCl with cooling, and the mixt. kept at room temp. 2 days, and neutralized to pH 7 with dil. NH4OH to give I (X = OH, Y = H2N), decomp. 327.degree.. Me2SO4 (3.4 ml.) added with shaking over 30 min. to 5 g. III in 100 ml. 0.35N NaOH gave I (X = MeS, Y = H2N) (IV), plates, m. 164-5.degree. (MeOH-ether-petr. ether). IV (5 g.) was heated in a bomb 72 hrs. at 155.degree. with 250 ml. EtOH satd. at 0.degree. with NH3 to give beige crystals of I (X = Y = H2N), m. 234-8.degree. (EtOH) (decompn.). These compds. are useful as antimetabolites of the isosteric **pyrimidine** derivs., microorganism **inhibitors**, and pharmaceutical intermediates.

L9 ANSWER 53 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1958:116355 CAPLUS
 DOCUMENT NUMBER: 52:116355
 ORIGINAL REFERENCE NO.: 52:20662c-d
 TITLE: The metabolism of pyrazolo[3,4-d]pyrimidines by the rat
 AUTHOR(S): Feigelson, Philip; Davidson, Jack D.
 CORPORATE SOURCE: Columbia Univ.
 SOURCE: Cancer Research (1958), 18, 226-8
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 GI For diagram(s), see printed CA Issue.
 AB The recent development of a new series of carcinostatic purine analogs, the pyrazolo[3,4-d]pyrimidines, led to their study as substrates and **inhibitors** of certain purine **oxidation** systems in vitro. In the present study, pyrazoloadenine was found to be oxidized in

the rat to pyrazoloisoguanine. The rat metabolizes pyrazolo[3,4-d] pyrimidines as follows:

L9 ANSWER 54 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1958:61603 CAPLUS
 DOCUMENT NUMBER: 52:61603
 ORIGINAL REFERENCE NO.: 52:11153i,11154a
 TITLE: Subtle interactions of cupric ion with nucleic acid and components
 AUTHOR(S): Frieden, Earl; Alles, Jeanne
 CORPORATE SOURCE: Florida State Univ., Tallahassee
 SOURCE: J. Biol. Chem. (1958), 230, 797-804
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB cf. C.A. 51, 11424a. Subtle interactions between Cu(II) ions and nucleic acids and their components were studied by their **inhibition** of the Cu(II)-catalyzed **oxidation** of ascorbate. The effectiveness of Cu(II) chelation was purine > purine nucleotide = ribonucleic acid-deoxyribonucleic acid > purine nucleoside > **pyrimidine** nucleotide. The deoxyribose derivs. were uniformly stronger Cu(II) chelators. The structure of these chelates and their possible biol. significance are discussed.

L9 ANSWER 55 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1957:86264 CAPLUS
 DOCUMENT NUMBER: 51:86264
 ORIGINAL REFERENCE NO.: 51:15661g-i
 TITLE: Pyrazolopyrimidines as **inhibitors** and substrates of xanthine oxidase
 AUTHOR(S): Feigelson, Philip; Davidson, J. D.; Robins, Roland K.
 CORPORATE SOURCE: Columbia Univ.
 SOURCE: J. Biol. Chem. (1957), 226, 993-1000
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB cf. C.A. 50, 16911i, 13036c. The **inhibitor** and substrate specificities of xanthine oxidase were studied by use of purine analogs, pyrazolo(3,4-d)**pyrimidines** (loc. cit.). Pyrazoloisoguanine competitively **inhibits** the enzyme; 50% **inhibition** occurs at 10-6M. Pyrazoloadenine is less **inhibitory**; 50% **inhibition** is seen at 10-4M. Methylation of the amino or ring N atoms still further decreases **inhibition**. Xanthine oxidase catalyzed the **oxidation** of pyrazoloadenine to pyrazoloisoguanine, which was characterized by paper chromatography, ion exchange, and spectrophotometry. The possible relation between the **inhibition** in vitro of xanthine oxidase and the carcinostatic activities of these compds. is discussed (cf. C.A. 50, 483b).

L9 ANSWER 56 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1957:57304 CAPLUS
 DOCUMENT NUMBER: 51:57304
 ORIGINAL REFERENCE NO.: 51:10642g-i,10643a
 TITLE: Studies on the influence of cobalt chloride on the growth of actinomycetes. I
 AUTHOR(S): Kojima, Hisao; Matsuki, Midori
 CORPORATE SOURCE: Tohoku Univ., Sendai
 SOURCE: Tohoku J. Agr. Research (1956), 7, 175-87
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Twelve strains of actinomycetes were cultured in Czapek's medium contg. 0, 2, or 8 .gamma. of CoCl₂·6H₂O (I)/ml. The presence of I in the media, at a concn. of 2 .gamma./ml. resulted in complete growth **inhibition** of 8 of these strains. When one of these strains (K300) was cultured in a glucose-bouillon medium it was found that concns. of I as high as

100. μ g./ml. were tolerated. The rate of glucose **oxidation** of this strain when cultured on Czapek's medium was **inhibited** by I. The **inhibitory** effect of Co on the glucose **oxidation** rate of the K300 strain cultured in the glucose-bouillon medium was greater than that observed in the organisms grown in the Czapek medium. A strain (346) which showed no growth **inhibition** when I was added to the media also showed less **inhibition** of the glucose **oxidation** rate in the presence of I. Casein hydrolyzate added to media which contained concns. of I as high as 12 μ g./ml. resulted in a reversal of the growth **inhibition**. **Pyrimidine** and purine bases were only slightly effective in reversing growth **inhibition**. When L-histidine or L-cysteine was added to the media contg. Co (80 μ g./ml.) the growth **inhibition** due to Co was reversed. **Inhibition** of the glucose **oxidation** rate by Co did not occur when these compds. were present initially. Preincubation of the organisms in media contg. L-cysteine resulted in nearly complete reduction of the Co **inhibition** of the glucose **oxidation** rate.

L9 ANSWER 57 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1956:16740 CAPLUS
 DOCUMENT NUMBER: 50:16740
 ORIGINAL REFERENCE NO.: 50:3540a-c
 TITLE: Effect of purine and **pyrimidine** analogs on enzyme induction in *Mycobacterium tuberculosis*
 AUTHOR(S): Ottey, Leo
 CORPORATE SOURCE: Duke Univ., Durham, NC
 SOURCE: J. Pharmacol. Exptl. Therap. (1955), 115, 339-42
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB Addn. to the medium of 6-mercaptopurine, 2,6-diaminopurine, 5-aminouracil, 5-methyl-2-thiouracil, 6-methyl-2-thiouracil, 2-thiocytosine, or 2-thiouracil **inhibited** the formation of adaptive enzymes for the **oxidation** of benzoic acid by *M. tuberculosis*; 2-thioorotic acid had no effect. The first 5 compds. and also 2-thioorotic acid **inhibited** formation of the adaptive enzymes for the **oxidation** of myo-inositol; 2-thiouracil had no effect here.

L9 ANSWER 58 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1955:73412 CAPLUS
 DOCUMENT NUMBER: 49:73412
 ORIGINAL REFERENCE NO.: 49:13900h-i,13901a-b
 TITLE: Action of some derivatives of **pyrimidine** in the **oxidation** of pyrocatechol
 AUTHOR(S): Mkhitaryan, V. G.; Shukuryan, S. G.; Avakimova, E. A.
 SOURCE: Doklady Akad. Nauk Armyan. S.S.R. (1953), 17 (No. 3), 81-5
 DOCUMENT TYPE: Journal
 LANGUAGE: Russian
 AB 4-Methyl-2-thiouracil (I) in phosphate buffer at pH 7.26 strongly **inhibits** the **oxidation** of pyrocatechol (II) either in the presence or the absence of Cu⁺⁺ or Fe⁺⁺⁺ ions. In phosphate buffer at pH 6.4 I alone or in the presence of Cu⁺⁺ ion strongly **inhibits** the **oxidation** of II, but it does not **inhibit** the **oxidation** in presence of Fe⁺⁺⁺ ion. 4-Methyl-uracil (III) in phosphate buffer at pH 7.23 weakly **inhibits** the **oxidation** of II, but it shows little influence on the **oxidation** in the presence of Fe⁺⁺⁺ and Cu⁺⁺ ions. In a phosphate buffer at pH 6.37 III does not **inhibit** the **oxidation** of II alone or in the presence of Fe⁺⁺⁺ ion, but it does **inhibit** in the presence of Cu⁺⁺ ion. The tests were carried out with II which had been twice-recrystd. from phosphate buffer at pH 7.2 and pH 6.4. The **oxidation** of II is measured manometrically by the quantity of O

consumed in a Warburg app. at 37.degree.. In the Warburg respirometer were used 3.3-ml. samples (7 mg. II in 1 ml. soln., 5 mg. I in 2.1 ml. of soln., and 0.2 ml. 30% KOH). In the tests with Cu and Fe ions (used as their sulfates), 3.1-ml. samples contg. 0.3 mg. Fe (0.06 ml.) or 0.003 mg. Cu (0.06 ml.) were employed.

L9 ANSWER 59 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1955:43135 CAPLUS

DOCUMENT NUMBER: 49:43135

ORIGINAL REFERENCE NO.: 49:8339a-e

TITLE: Second type of bacterial thiaminase

AUTHOR(S): Fujita, Akiji; Nose, Yoshitsugu; Kuratani, Kazuo

CORPORATE SOURCE: Kyoto Prefectural Univ.

SOURCE: J. Vitaminol. (Japan) (1954), 1, 1-7

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB The properties of thiaminase II (I) of *Bacillus aneurinolyticus* were different from thiaminase from *Bacillus thiaminolyticus*, shellfish, fish, and plant sources, called thiaminase I (II). The effects of various amines (III) on I (pH 7, 60.degree., with a final concn. of III of 10-3M) by use of the same method as was used on II by F., et al. (C.A. 46, 12281h), showed I not to be activated and in many cases inhibited by III, contrary to the findings on II. With pyridine and quinoline the fluorescent substance (pyrichrome and quinochrome) did not appear after ferricyanide oxidation, showing the lack of the base-exchange reaction. Enzymic action of I on thiamine produced pyrimidinemethanol (IV) and the thiazole moiety (V), whereas II never gave IV and the fate of the pyrimidine moiety could not be traced in this case by the Dragendorff reagent. Since I was not activated by various III, the base-exchange reaction was not expected. However, the formation of pyrimidinemethylaniline (VI) by I in the presence of thiamine and aniline was shown as in the case of II. Similar base-exchange reactions with other III did not take place and only the formation of IV was demonstrated. A study of various molar ratios of aniline to thiamine in the formation of VI showed that IV formed in low aniline concn. while VI formed in aniline concn. higher than twice that of thiamine. It was observed that thiamine was formed as the reverse reaction of thiaminase by incubating II with the base-exchanged pyrimidine deriv. and the thiazole moiety of thiamine (loc. cit.). I produced a reverse reaction when incubated with VI, contrary to the case of II. It seems probable from the above findings to assume that I itself does not catalyze the base-exchange reaction, but that a second enzyme accompanying it catalyzes the reaction which makes the reaction appear to be a base exchange. Furthermore, since the base-exchange reaction does not take place with other III, but aniline, makes the assumption of a special enzyme probable.

L9 ANSWER 60 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1954:43494 CAPLUS

DOCUMENT NUMBER: 48:43494

ORIGINAL REFERENCE NO.: 48:7790f-h

TITLE: Oxidation of ascorbic acid by Terramycin

AUTHOR(S): Dudani, A. T.; Krishnamurti, C. R.

CORPORATE SOURCE: Central Drug Research Inst., Lucknow, India

SOURCE: Biochim. et Biophys. Acta (1954), 13, 505-9

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB Terramycin (I) (and dihydrostreptomycin to a very much lesser degree) accelerated the rate of oxidation of ascorbic acid (II).

Aureomycin, neomycin, and penicillin, on the other hand, inhibited

oxidation, penicillin G exerting the strongest effect. I did not

cause oxidation of any of a large no. of reducing agents,

suggesting that the action of I is specific for II. The effect of I was

proven not to be caused by Cu impurity. The catalytic activity of I was found to be thermostable, to not affect either antibacterial activity or fluorescence of I in the ultraviolet, to have a pH optimum of 8, and to be present in amounts as low as 1 .gamma. of I. Penicillin G and 8-hydroxyquinoline inhibited the activity of I, but a variety of purines, pyrimidines, amino acids, vitamins, HCN, etc. had no effect. Many of the substances, however, inhibited Cu catalysis.

L9 ANSWER 61 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1954:42669 CAPLUS
 DOCUMENT NUMBER: 48:42669
 ORIGINAL REFERENCE NO.: 48:7670a-b
 TITLE: The inhibitors of the oxidation of L-ascorbic acid. The mechanism of action of thiamine
 AUTHOR(S): Gero, Etienne
 SOURCE: Compt. rend. (1954), 238, 959-61
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB cf. C.A. 47, 2781c. Cuprothiamine contains 2 atoms of Cu per mol. of thiamine. The thiazole nucleus (I) strongly inhibits the oxidation of L-ascorbic acid by Cu++ ions. The pyrimidine nucleus (II), an inhibitor by itself, diminishes the action of I when I is assoc'd. with II in the same mol.

L9 ANSWER 62 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1954:32707 CAPLUS
 DOCUMENT NUMBER: 48:32707
 ORIGINAL REFERENCE NO.: 48:5890h-i,5891a
 TITLE: The action of 4-methyluracil and 4-methyl-2-thiouracil on the oxidation process of ascorbic acid
 AUTHOR(S): Mkhitaryan, V. G.; Avakimova, E. A.; Shchukuryan, S. G.
 CORPORATE SOURCE: Sci. Research Inst. Roentgenol. and Oncol., Ministry Health Armenian S.S.R., Erevan
 SOURCE: Voprosy Pitaniya (1953), 12(No. 4), 23-8
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB The oxidation of ascorbic acid (I) solns. was detd. manometrically in a Warburg respirometer at 37.degree. and by indophenol titration. 4-Methyl-2-thiouracil (II) inhibits the oxidation of I more effectively in phosphate buffer at 6.24 than at pH 7.23. This compd. inhibits the oxidation of I in the presence of Cu ions at pH 6.24 and is as strong an antioxidant as uric acid. The antioxidative action of II in the presence of Fe ions is much weaker. 4-Methyluracil alone shows some inhibitory action on the oxidation process of I at pH 6.24, but does not depress the oxidation in the presence of Fe and Cu ions at this pH. II shows stronger inhibitory action on the oxidation process of I than 4-methyluracil. The mechanism of II action on the oxidation of I is believed to be due to the action of the pyrimidine ring and to the bivalent S.

L9 ANSWER 63 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1952:36146 CAPLUS
 DOCUMENT NUMBER: 46:36146
 ORIGINAL REFERENCE NO.: 46:6172c-g
 TITLE: The structure of nucleic acids. II. Investigation of pentose nucleic acid and enzyme-resistant residues
 AUTHOR(S): Cavalieri, Liebe F.; Kerr, Stanley E.; Angelos, Alice
 CORPORATE SOURCE: Sloan-Kettering Inst., New York, NY
 SOURCE: J. Am. Chem. Soc. (1951), 73, 2567-78
 DOCUMENT TYPE: Journal

LANGUAGE: Unavailable
 AB cf. C.A. 46, 6089b. The interaction of rosaniline with various fractions of yeast nucleic acid and pentose nucleic acid (PNA) from beef pancreas was studied. The binding sites involve the phosphoric acid groups, about 13% of which are available for binding. On the basis of the similar intrinsic binding consts. and n values (no. of available sites), it is suggested that a similar backbone structure exists among the nucleic acid samples studied. The interaction of rosaniline with the ribonuclease-resistant fractions of yeast PNA was also studied. The binding capacity of the n1-type site (bivalent ion) of the resistant fraction is slightly greater than that of the parent PNA, owing to a decrease in steric **inhibition**. In the case of the ribonuclease-phosphatase-treated nucleic acid, the results of the binding process suggest that only one type of site is involved, which corresponds to the univalent anion type. Correlation of the exptl. pH titration curves with theoretical curves constructed from the known compn. of various samples of nucleic acid, a ribonuclease-resistant fraction and a ribonuclease-phosphatase-resistant fraction indicates that some of the OH groups of guanine and/or uracil are unavailable for titration and may be covalently bound in a phosphate-type bond. In the case of the ribonuclease-phosphatase-treated nucleic acid, purine and **pyrimidine** analyses, periodate **oxidation** titers and ion-exchange analysis provide addnl. evidence for such a covalent linkage. Both PNA and the ribonuclease-resistant fraction were subjected to periodate **oxidation**, and it appears that a D-riboside phosphate other than the 2' or 3' exists in the resistant fraction. Ultraviolet and infrared absorption spectra of both treated and untreated nucleic acids are similar and cannot be used effectively as a means of identification. X-ray powder patterns suggest that some of the residues remaining after the action of ribonuclease and acid-phosphatase are partially cryst.

L9 ANSWER 64 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1952:738 CAPLUS
 DOCUMENT NUMBER: 46:738
 ORIGINAL REFERENCE NO.: 46:151g-h
 TITLE: **Inhibition** of the oxidases of *Agaricus campestris*
 AUTHOR(S): Voinovitch, Igor
 CORPORATE SOURCE: Inst. natl. conserve, Paris
 SOURCE: Bull. soc. chim. biol. (1951), 33, 337-46
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable

AB cf. C.A. 44, 3540e, 8980b. The **oxidation** in presence of air of pyrogallol and tyrosine by a partially purified soln. of the polyphenol oxidases of *A. campestris* is **inhibited** more strongly by thiamine than by ascorbic acid. The thiazole moiety of thiamine **inhibits** the **oxidation** of tyrosine and the **pyrimidine** moiety **inhibits** the **oxidation** of pyrogallol. Nicotinic acid **inhibits** the **oxidation** of pyrogallol by the enzyme prepn.; nicotinamide does not. In all the above cases SO₂ augments the **inhibiting** action. Cysteine, and glutathione plus SO₂, weakly **inhibit** the **oxidation** of pyrogallol. Cystine is inert. Cysteine increases the activity of the succinic dehydrogenase of the mushroom; glutathione is much less active.

L9 ANSWER 65 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1951:19279 CAPLUS
 DOCUMENT NUMBER: 45:19279
 ORIGINAL REFERENCE NO.: 45:3431b-c
 TITLE: **Inhibitory** effect of caffeine and methylene blue on amine oxidase
 AUTHOR(S): Ota, Yukito
 SOURCE: J. Biochem. (Japan) (1950), 37, 289-99

DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB The endogenous respiration of guinea-pig brain pulp at pH 7.6 was inhibited 50% by M/500 tyramine (I), and 20% by M/50 caffeine (II); the inhibition by I was halved by the simultaneous addn. of M/50 II. Amine oxidase (III) was prep'd. by extg. guinea-pig liver with buffer at pH 7.0, followed by dialysis for 5 hrs. The oxidation of I by III was inhibited by II and by methylene blue. Among the derivs. of purine, **pyrimidine**, and some dyes, the mols. having N-methyl or amino group showed inhibition of III.

L9 ANSWER 66 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1950:33621 CAPLUS
 DOCUMENT NUMBER: 44:33621
 ORIGINAL REFERENCE NO.: 44:6461i,6462a-h
 TITLE: Role of copper in the Nadi reaction, and nonprotein models of oxidases and catalase
 AUTHOR(S): Pongratz, Edmond
 CORPORATE SOURCE: Univ., Geneve, Switz.
 SOURCE: Helv. Chim. Acta (1950), 33, 410-17
 DOCUMENT TYPE: Journal
 LANGUAGE: French
 AB To 10 cc. of pH 7.3 phosphate buffer were added 50 .gamma. Cu⁺⁺ and approx. 0.2 cc. of Nadi mixt. (equal amts. of 0.05 M 1-naphthol in EtOH and either 0.05 M p-C₆H₄(NH₂)₂ (I) or the N, N-di-Me deriv. of I in EtOH). The color reaction characteristic of cytochrome oxidase (red or blue, resp.) was given under these conditions, but not when, instead of Cu⁺⁺, any of 33 other common cations or anions was used. AgNO₃ gave an instantaneous gray violet color with the Nadi reagent, and NH₄ vanadate weakly catalyzed the oxidation of Nadi. The Cu-catalyzed reaction was inhibited by Cu-pptg. reagents such as H₂S, sol. sulfides, KSCN, and (COOH)₂; substances forming complexes with Cu such as dithiooxamide, salicylaldoxime, 8-hydroxyquinoline, cupferron, benzoin oxime, Na diethyldithiocarbamate, dithizone, thiourea, thiouracil, glycine, alanine, D-serine, aspartic acid, HCN, and CO; and oxidizable substances such as ascorbic acid, cysteine, glutathione, and Fe⁺⁺, which compete with the Nadi reagent and thus forestall its oxidation. NH₂OH did not fall into this last group, and typical inhibitors of dehydrogenases (ICH₂COOH, urethan, narcotics, and NaF) did not affect the Cu-catalyzed Nadi reaction. The inhibitors cited are, in general, those of true oxidases. The catalytic effect of Cu was increased by many org. compds. contg. tertiary or quaternary N. The most active found were NH₃, pyridine, iminazole, **pyrimidine**, and many of their derivs., such as Me₃N, chloramine, choline, nicotinic acid, nicotinamide, coramine, aneurine, biuret, uracil, barbiturates, and guanine. CN⁻ acted as an activator when its concn. was less than 1 mole per mole Cu. Its effect was greater when it was added to the Cu after, rather than before, the substrate. The effect of the activators is due to augmentation of the oxidation potential of the Cu. As the pH of reaction mixts. contg. Nadi was raised from pH 5, the rate of the Cu-catalyzed oxidation of the dimethyl-I at 18.degree. increased until pH 7.2, when it leveled off. The rate of oxidation catalyzed by Cu-pyridine kept increasing up to a pH of more than 8. Raising the temp. increased the catalytic effect of Cu complexes, until at a temp. of 80-90.degree. a max. was reached. The solv. of O then became a limiting factor. Cu⁺⁺, especially in the form of org. complexes, exhibited an oxidase-like action. Cu-iminazole was particularly effective, and oxidized hydroquinone (II), pyrogallol (III), and tincture of guaiac rapidly; and guaiacol (IV), catechol (V), resorcinol, orcinol, protocatechuic acid, adrenaline, 1-naphthol, and similar compds. more slowly. Tyrosine was not attacked, but dihydroxyphenylalanine was oxidized to melanin. Monophenols, p-cresol (VI) and a mixt. of VI with glycine were not attacked. In the presence of H₂O₂, Cu-iminazole

catalyzed the rapid oxidation of II, III, IV, V, phloroglucinol, o-, m-, and p-VI, m- and p-C₆H₄(NH₂)₂, benzidine, and related compds. The decompn. of H₂O₂ by Cu(NH₃)₄⁺⁺ was much greater than that by Cu⁺⁺, and was enhanced by adsorption of the catalyst on chalk. Mn⁺⁺-nicotine complex adsorbed on chalk was also effective. In addn. to the value of these complexes as models of enzymes, they may prove useful for Cu analysis, because of the specificity of the reaction of Cu with the Nadi reagent.

L9 ANSWER 67 OF 73 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1949:17545 CAPLUS

DOCUMENT NUMBER: 43:17545

ORIGINAL REFERENCE NO.: 43:3426g-i,3427a-i,3428a-c

TITLE: Some heterocyclic analogs of stilbenes

AUTHOR(S): Brown, Daniel M.; Kon, George A. R.

SOURCE: J. Chem. Soc. (1948) 2147-54

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB Derivs. of 4-aminostilbene are known to be carcinogenic and also to exercise an inhibitory effect on the development of transplanted tumors in the rat. 4-Methylpyrimidine (1 g.), 1.5 g. 4-Me₂NC₆H₄CHO (I), and 0.5 g. ZnCl₂, heated 1.5 hrs. at 165.degree., give 1.2 g. 4-(4-dimethylaminostyryl)pyrimidine, yellow, m. 179.degree. (picrate, purple, m. 195-6.degree., forms black needles at 175.degree.). 2-Hydroxy-4,6-dimethylpyrimidine (II) and 1 mol. I give 2-hydroxy-4,6-bis(4-dimethylaminostyryl)pyrimidine (III), purple, with 0.5 mol. H₂O, decomp. 316-18.degree. [from (HOC₂H₄)₂O]; the residue from MeOCH₂CH₂OH gives the monostyryl deriv. (IV), scarlet, m. 253.degree. (decompn.) (Stark and Bogemann, C.A. 4, 2466). I (1.5 g.) and 1.5 g. II.HCl in 20 cc. EtOH and 10 cc. H₂O, boiled 4 hrs., give a mixt. of III and IV; 1 drop concd. HCl gives the same mixt. II (1.1 g.), 3 g. I, and 10 drops piperidine in 100 cc. EtOH, refluxed 48 hrs., give III. The crude mixt. of III and IV (2 g.), refluxed 2 hrs. with 10 cc. POCl₃, gives 1.2 g. 2-chloro-4-(4-dimethylaminostyryl)-6-methylpyrimidine (V), yellow, m. 176-7.degree., and 0.1 g. 2-chloro-4,6-bis(4-dimethylaminostyryl)pyrimidine (VI), red, m. 223-4.degree.; these were sepd. by chromatography on Al₂O₃. VI (0.1 g.) and 1 cc. piperidine refluxed 3 min. give 0.025 g. of the 2-(1-piperidyl) compd., yellow, m. 223-4.degree.. V (0.52 g.) gives 0.6 g. of the 2-(1-piperidyl) compd. (VII), light yellow, m. 168-9.degree.; 2-(4-morpholinyl) analog (VIIA), pale yellow, m. 155.degree., 100%; 2-cyclohexylamino analog, bright yellow, m. 142-3.degree., 82%; 2-(2-diethylaminoethylamino) analog, yellow, m. 80-1.degree., 65%; 2-diethylamino analog, yellow (from petr. ether) or greenish yellow (from aq. alc.), m. 121-2.degree., 74%; 2-[bis(2-hydroxyethyl)amino] analog, bright yellow, m. 116.degree.. VII (1 g.) in 50 cc. EtOH, hydrogenated over 2% Pd-SrCO₃ at room temp./atm. pressure, gives 0.8 g. 2-(1-piperidyl)-4-[2-(4-dimethylaminophenyl)ethyl]-6-methylpyrimidine (VIII), m. 70.degree.. V (1 g.) and 0.085 g. Na in 25 cc. EtOH and 10 cc. C₆H₆, refluxed 1.5 hrs. on a steam bath, give 0.8 g. 2-ethoxy-4-(4-dimethylaminostyryl)-6-methylpyrimidine, orange, m. 120.degree.. The Cl in V could not be removed by refluxing with Zn in aq. dioxane; catalytic reduction gives the 4-[2-(4-dimethylaminophenyl)ethyl] deriv., m. 59-60.degree.; with piperidine it yields VIII. 2-Hydroxy-4-styryl-6-methylpyrimidine. (1.1 g.) and 6 g. POCl₃, refluxed 1 hr., give 0.35 g. of the 2-Cl compd., m. 95.degree.; 2-(1-piperidyl) compd., light yellow, m. 94.degree.. 2-Hydroxy-4,6-distyrylpyrimidine yields the 2-Cl compd., m. 177-8.degree.; 2-(1-piperidyl) compd., light yellow, m. 133.degree.. 2-(1-Piperidyl)-4,6-dimethylpyrimidine (IX) m. 60-1.degree.. 2,6-Di-1-piperidyl-4-methylpyrimidine (X) m. 118.degree.. IX and X failed to react with I under a variety of conditions. 2-Amino-4,6-dimethylpyrimidine (5 g.), refluxed 5 min. with 15 cc. Ac₂O, treated with 6 g. I in 5 cc. Ac₂O, and refluxed 1 hr., gives 0.2 g. 2-acetamido-4-(4-dimethylaminostyryl)-6-methylpyrimidine, yellow, m. 218-19.degree.. VII and VIIA possess considerable growth-

inhibiting action. 6-Quinolinecarboxaldehyde (XI) (5 g.), 5.8 g. 4-O2NC6H4CH2CO2H, and 2 cc. piperidine, heated 1.5 hrs. at 130-40.degree., give 3 g. 6-(4-nitrostyryl)quinoline (XII), yellow, m. 199-200.degree.; reduction of 2 g. XII with 16 g. SnCl2 in 40 cc. AcOH satd. with HCl (stirred several hrs. at room temp. and heated 4 hrs. on the steam bath) gives 0.87 g. 6-(4-aminostyryl)quinoline (XIII), yellow, m. 214-15.degree.. The 8-isomer of XI (6.75 g.) yields 2.3 g. of the 8-isomer of XII, yellow, m. 171.degree.; reduction of 0.5 g. gives 0.38 g. of the 8-isomer of XIII, yellow, m. 156.degree.. XI (3.64 g.) in 25 cc. C6H6, added to PhCH2MgCl (3.3 g. PhCH2Cl) in 50 cc. ether and refluxed 2 hrs., gives only 0.25 g. 2-phenyl-1-(6-quinolyl)ethanol, m. 129.5-30.degree.. PhCH2CO2Na (0.9 g.), 0.9 g. XI, 5 cc. Ac2O, and 0.2 g. ZnCl2, heated 3 hrs. at 160.degree., give 1.4 g. α -phenyl-6-quinolineacrylic acid, m. 265.degree.; it could not be decarboxylated. A Skraup synthesis with XI and 4-aminostilbene gave no recognizable product. The diazo compd. from 0.5 g. XIII, decompd. with H3PO2, gives only 10 mg. 6-styrylquinoline, m. 119.degree.. SnCl2 reduction of 6-nitro-6-styrylquinoline yields the 6-NH2 compd., brown, m. 198-9.degree.; Ac deriv., with 1 mol. CHCl3, m. 193.degree.. 6-Nitroquinaldine (XIV) (5 g.), 4 g. I, and 0.2 g. ZnCl2, heated 0.5 hr. at 160.degree., give 7.6 g. 6-nitro-2-(4-dimethylaminostyryl)quinoline, deep purple, m. 248-9.degree., 2 g. of which with 10 g. SnCl2 and 15 cc. fuming HCl, heated 1 hr. on the steam bath, give 1.15 g. of the 6-NH2 compd., brown, m. 251-2.degree.; Ac deriv., orange-yellow, m. 241-2.degree.; Sn and HCl give 2-(2-phenylethyl) quinoline. XIV (2.1 g.), 1.7 g. 4-O2NC6H4CHO, and a little ZnCl2, heated at 170.degree., give 6-nitro-2-(4-nitrostyryl)quinoline, yellow, m. 278.degree.; the 6-NH2 deriv. m. 242-3.degree.; these amines are very sensitive to aerial oxidation. 1-Methyltetrahydroquinoline (10 g.), added dropwise to 10.5 g. POCl3 and 9.2 g. PhNMeCHO in 10 cc. C6H6 and kept overnight, gives 5.5 g. 6-formyl-1-methyl-1,2,3,4-tetrahydroquinoline (XV), b15 219-21.degree., m. 28-9.degree.; 1.53 g. XV in 25 cc. C6H6, added to PhCH2MgCl (1.23 g. PhCH2Cl) in 30 cc. ether, refluxed 45 min., and the reaction product in 25 cc. C6H6 refluxed 45 min. with 2 g. P2O5, gives 0.6 g. 6-styryl-1-methyl-1,2,3,4-tetrahydroquinoline, m. 93-4.degree.. 2-Methylthiazole (1 cc.), 1.5 g. I, and 0.5 g. ZnCl2, heated 14 hrs. at 160-70.degree., give 10 mg. 2-(4-dimethylaminostyryl)thiazole, yellow, m. 124.degree.. 2-Methylnaphtho[1,2]thiazole (0.5 g.), 0.4 g. I, and 0.5 g. ZnCl2, heated 1.5 hrs. at 160-80.degree., give 0.46 g. of the 2-(4-dimethylaminostyryl) deriv., yellow, m. 170-1.degree.. 2-Methylbenzothiazole (1 g.), 0.8 cc. I, and 2 drops concd. HCl, heated overnight at 100.degree., give 0.46 g. 2-styrylbenzothiazole, m. 112.degree.. 2-Methylbenzoxazole (XVI) gives an HCl salt, m. 154.degree.. XVI (2 cc.), 2 cc. BzH, and 1 g. anhyd. ZnCl2, heated 6 hrs. at 160.degree., give 1 g. 2-styrylbenzoxazole m. 81-2.degree. (picrate, yellow, m. 163-4.degree.); 2-(4-dimethylaminostyryl) compd., yellow, m. 174-5.degree.. 1-Methylphthalazine (2.5 g.), 2.75 g. I, and 0.75 g. ZnCl2, heated 2 hrs. at 160.degree., give 0.39 g. 1-(4-dimethylaminostyryl)phthalazine, orange, m. 186-7.degree.. Furfuraldehyde (5 g.), 9 g. 4-O2NC6H4CH2CO2H, and 1 cc. piperidine, heated 5 hrs. at 130-40.degree., give 2 g. 2-(2-nitrostyryl)furan (XVII), orange, m. 130-1.degree.; the addn. of p-O2NC6H4N2Cl to 2-furanacrylic acid in Me2CO, followed by AcONa and CuCl2, gives XVII. Catalytic reduction of XVII over Raney Ni gives the azoxy compd., orange, m. 231-2.degree. (decompn.); reduction with Zn and NH4Cl in EtOH gives 2-(4-aminostyryl)furan, m. 104.degree.; Ac deriv. m. 201-2.degree.; reduction under acid conditions gives resinous products.

properties of thiopterins
 AUTHOR(S) : Polonovski, Michel; Guinand, Sylvanie; Pesson, Marcel;
 Vieillefosse, Roger
 SOURCE : Bull. soc. chim. (1945), 12, 924-9
 DOCUMENT TYPE : Journal
 LANGUAGE : Unavailable
 GI For diagram(s), see printed CA Issue.
 AB cf. C.A. 40, 874.6. Thiopterins of type I do not fluoresce while those of type II show an intense blue or green fluorescence similar to pterin derivs. of types III and IV. When I are oxidized with H₂O₂, the S is replaced by O, and the **oxidation** product exhibits strong fluorescence. In II and in IV, 3 conjugated double bonds are present in the **pyrimidine** ring, and as soon as these double bonds disappear, as in I, the fluorescence also disappears. To study this phenomenon further, the ultraviolet absorption spectra of I (R, R' = H), its **oxidation** product (V), II (R, R' = H), IV (R, R' = H), 2-thio-6-oxo-8,9-diphenyl-1,6,2,3-tetrahydropteridine (VI) (I, R, R' = Ph), the **oxidation** product (VII) of VI, 2-ethylmercapto-6-hydroxy-8,9-diphenylpteridine (VIII) (II, R, R' = Ph), and 2,6-dihydroxy-8,9-diphenylpteridine (IX) (IV, R, R' = Ph) are detd. I and VI show absorption max. at 3065 and 3180 Å. (Hg light). On **oxidation** of I and VI, the absorption max. shift to the shorter wave lengths, those of V, II, and IV lying at 2700, 2715, and 2700 Å., resp., those of VII, VIII, and IX at 2870, 2840, and 2870, resp. The fluorescence of the compds. with R, R' = H is very strong in alk. soln. and becomes very weak in acid soln. due to the change from the lactim form into the lactam form under the influence of the pH. Since some S compds. exhibit an antifluorescence activity (cf. Perrin, C.A. 21, 3012), it is possible that a fluorescence which may originate from an equil. between the thione form (X) and the thiol form (XI) in I may be prevented by the anti-fluorescence properties of X. The effect of thiourea (XII) upon the fluorescence of II (R, R' = H) and III is studied, and it is found that XII, unlike urea, exhibits a strong antifluorescence activity. Because I contains the same grouping as is present in XII, P. et al. assume that I also acts as a fluorescence **inhibitor**. Addn. of I to a soln. of II or III also decreases the fluorescence of the latter to an even greater extent than the addn. of XII.

L9 ANSWER 69 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1942:2977 CAPLUS
 DOCUMENT NUMBER: 36:2977
 ORIGINAL REFERENCE NO.: 36:508b-d
 TITLE: Using *Phycomyces blakesleeanus* in the determination of vitamin B1
 AUTHOR(S): Malm, Max; Lundeen, Harry
 SOURCE: Svensk Kem. Tid. (1941), 53, 246-64
 DOCUMENT TYPE: Journal
 LANGUAGE: German
 AB cf. Sinclair, C. A. 33, 4625.8. Vitamin B1 is detd. by the increase in dry wt. of the mycelium of *Phycomyces* in glucose-asparagine substrate to which the sample has been added. Casein hydrolyzate, age of culture from which spores are taken, and light are **inhibiting** factors of growth but do not interfere sufficiently to vitiate the method. Equiv. concns. B1, cocarboxylase, B1 orthophosphate, disulfide and **pyrimidine** + thiazoleacetate from B1 have the same effect on the growth. The disulfide of B1 gives a slower growth rate than B1 and the rate increases with the concn. of the disulfide. **Pyrimidine** sulfonate + thiazole, nicotinamide, adermin, .beta.-alanine, cystine, glutathione and heparin do not promote the growth of the fungus. Thiochrome from B1 by K₃FeC₆N₆ **oxidation** does not promote growth. In an ideal substrate the increased mycelium wt. is directly proportional to the concn. B1. A method of calcn. is given for substrates having activating or **inhibitory** factors. Sinclair's correction

factor is not accepted.

L9 ANSWER 70 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1941:48047 CAPLUS
 DOCUMENT NUMBER: 35:48047
 ORIGINAL REFERENCE NO.: 35:7455g-i,7456a-c
 TITLE: Vitamin B1 and bacterial oxidations. I. Dependence of acetic acid **oxidation** on vitamin B1
 AUTHOR(S): Quastel, J. H.; Webley, D. M.
 SOURCE: Biochem. J. (1941), 35, 192-206
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB cf. C. A. 34, 791.5. The presence of vitamin B1 (I) at low concns. (10.-7 M) greatly increased the rate of **oxidation** of AcOH by I-deficient propionic acid bacteria. The molar ratio of O consumed to AcOH used in the presence and absence of I was approx. 2.0; this shows complete **oxidation** of AcOH. HC₂O₂H, AcOH, EtCO₂H and PrCO₂H were oxidized at the same rate by I-deficient propionic acid bacteria. The addn. of I caused little change in the rate of **oxidation** of HC₂O₂H and PrCO₂H, but increased the rate of **oxidation** of EtCO₂H. The increase was much less than with AcOH. Pyruvic acid (II) accumulated during the **oxidation** of glucose (III), lactic acid (IV), glycerol (V) and PrCO₂H by deficient bacteria. Its amt. was greatly decreased in the presence of I. The rate of utilization of AcOH by these organisms with added I was approx. equal to that of II. The presence of IV, succinic acid (VI), fumaric acid (VII) or V inhibited the utilization of II by propionic acid bacteria in the presence of I. EtCO₂H caused some **inhibition** but AcOH had no effect. The presence of I greatly stimulated the rates of respiration by deficient bacteria with the following substrates: II, IV, VI, VII, III, V. Definite but less marked effects were shown by I in the oxidations of l-malic acid, l-glutamic acid, dl-alanine, glycolic acid, glycine and fructose. Little or no **oxidation** occurred in any case with oxalic acid, citric acid or .beta.-hydroxybutyric acid. The **oxidation** of .alpha.-glycerophosphoric and hexosediphosphoric acids was not affected by I. The stimulant action of I was attributed mainly to its effect on the **oxidation** of II formed as an intermediate. The effect of I on the **oxidation** of AcOH could not be explained in this way. EtOH was oxidized by I-deficient propionic acid bacteria, the rate of **oxidation** being greatly increased by the addn. of I. AcOH which accumulated during the **oxidation** of EtOH was greatly reduced when I was present, probably accounting for the increased rate under such conditions. PrOH was vigorously oxidized by deficient bacteria, but the rate was not affected greatly by I. MeOH was only slightly oxidized in both instances. The **pyrimidine** and thiazole components of I were separately unable to catalyze the **oxidation** of AcON by propionic acid bacteria.

L9 ANSWER 71 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1938:39030 CAPLUS
 DOCUMENT NUMBER: 32:39030
 ORIGINAL REFERENCE NO.: 32:5437a-b
 TITLE: Aneurin (vitamin B1) and pyruvate metabolism by *Staphylococcus aureus*
 AUTHOR(S): Hills, Geo. M.
 SOURCE: Biochem. J. (1938), 32, 383-91
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB The effect of aneurin (I) on the metabolism of *Staph. aureus* occurred at very low concns. and could only be observed in organisms which received an inadequate supply of the vitamin during growth. Both the **pyrimidine** and the thiazole rings of I were necessary for the normal metabolism of pyruvic acid (II) by *staphylococcus* both under

aerobic and anaerobic conditions. In the metabolism of lactate (III) and II the presence of I was necessary for the reaction which involved the dismutation of I into lactic acid, AcOH and CO₂. In the absence of I, there was an accumulation of II which acted as an **inhibitor** on the **oxidation** of III.

L9 ANSWER 72 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1937:38292 CAPLUS
 DOCUMENT NUMBER: 31:38292
 ORIGINAL REFERENCE NO.: 31:5395c-f
 TITLE: Decomposition of histidine and of other imidazoles by ascorbic acid
 AUTHOR(S): Edlbacher, S.; v. Segesser, A.
 SOURCE: Biochem. Z. (1937), 290, 370-7
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB Histidine unlike other amino acids is absolutely resistant to oxidative deamination but its imidazole ring is split open hydrolytically by a histidase present in the liver. There is a close analogy to the related arginine which is likewise hydrolyzed to ornithine and urea by liver arginase. The histidase gives a labile product contg. one N atom less, which on alkalinating with NaOH gives off a second N atom as NH₃, and acts equally well in the presence or absence of O₂ and is unaffected by KCN. When histidine is incubated with ascorbic acid at pH 7 in the presence of a trace of Fe₂(SO₄)₃ or of hemin similar products are formed, only in this case there is an oxidative reaction (depends on presence of O₂ and is inhibited by CN). Expts. with various biologically important substances show that only the simple imidazole derivs. are destroyed by this reaction whereas hypoxanthine and adenine with the imidazole ring bound to a **pyrimidine** ring are at least partially deaminated. The question whether other reduction-**oxidation** substances besides ascorbic acid can be used is being investigated. The biol. significance of the place occupied by ascorbic acid in the **oxidation-reduction** system of the cell is discussed.

L9 ANSWER 73 OF 73 CAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1935:19870 CAPLUS
 DOCUMENT NUMBER: 29:19870
 ORIGINAL REFERENCE NO.: 29:2554a-c
 TITLE: Experimental studies on nuclein metabolism. XXXVII.
 Nucleosidase
 AUTHOR(S): Klein, Willibald
 SOURCE: Z. physiol. Chem. (1935), 231, 125-48
 DOCUMENT TYPE: Journal
 LANGUAGE: Unavailable
 AB cf. C. A. 28, 4079.9. A micro method for detg. hydrolysis of purine desoxyribosides by nucleosidase is based on removal of protein by Pb(NO₃)₂, addn. of 0.02 N I and 0.1 N NaOH, acidification with N H₂SO₄ and titration of excess I with 0.01 N Na₂S₂O₃ and starch indicator. The intact nucleosides are not attacked but the liberated desoxypentose undergoes **oxidation**. Nucleosidase is obtained by drying frozen organs, adsorbing the ext. on Al(OH)₃ and eluting with Na₂HASO₄, the most active preps. being obtained from spleen, lung, liver and heart muscle. AsO₄---, and to a smaller extent PO₄---, is the activator. The enzyme is sp. for the purine nucleosides and does not attack **pyrimidine** nucleosides, nucleotides or nucleic acids. Guanine and hypoxanthine are strong **inhibitors**, adenine is less so and desoxyribose very slightly so, while the **pyrimidine** nucleosides and the nucleotides have no effect. Purine nucleosidases from various organs are identical, as are also the riboside and desoxyriboside nucleosidases. In addn. to purine nucleosidase there is a sp. **pyrimidine** nucleosidase. This is more abundant in kidney than in spleen and red marrow.

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(FILE 'HOME' ENTERED AT 17:22:47 ON 02 SEP 2002)

FILE 'REGISTRY' ENTERED AT 17:22:55 ON 02 SEP 2002

L1 STRUCTURE UPLOADED
L2 3 S L1
L3 330 S L1 FUL

FILE 'CPLUS' ENTERED AT 17:23:39 ON 02 SEP 2002

L4 323 S L3
L5 9814 S ELECTRON DONATING
L6 1 S L4 AND L5
L7 24310 S INHIBIT? AND OXIDATION
L8 0 S L4 AND L7
L9 73 S L7 AND PYRIMIDIN?
L10 14 S L9 AND HYDROXY
L11 0 S L7 AND (HYDROXY SAME PYRIMIDIN?)
L12 0 S L7 AND (HYDROXY PYRIMIDIN?)

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